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- Amyloidosis
- Hemochromatosis

- Sepsis
- TPN

} ICU patients

Alk. Phosphatase ↑

- Isolated
  - ↓
  - Check GGT level
  - ↓ (N)
  - Evaluate extrahepatic causes
- GGT ↑ cholestatic
  - ↓
  - USG
  - ↓ **Dilated ducts**
    - Sub Acute / chronic
      - ↓
      - Tumour
      - Biliary stricture
    - Acute
      - ↓
      - Stone

Non dilated ducts

- Acute
  - ↓
  - Drugs
  - Sepsis
- Non Acute
  - Antimitochondrial Ab
    - ↓
    - PBC
  - HRCP - PSC.
  - Drugs, liver biopsy.

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Alk Phosphase – ↓

- Wilson's disease.

Rare
- Hypothyroidism
- Pernicious Anemia
- Zinc deficiency.

Bilirubin

Heme
↓
Protoporphyrin
↓
Biliverdin
↓
Bilirubin
(unconjugated)
↓
binds c̄ albumin (not water soluble)
↓
liver
↓ glucuronyl conjugation
Conjugated bilirubin
(water soluble)
↓
Bile duct → Intestine

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Intestine

Urobilinogen — Stercobilinogen

Total bilirubin → 1.2 mg/dL

Unconj. bilirubin → 0.9 mg/dL (Indirect)

Conjugated " → 0.8 mg/dL (Direct)

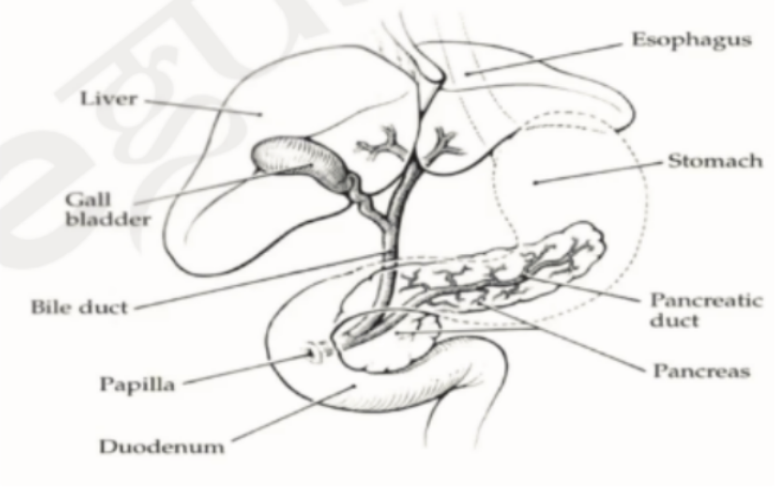
Total Bilirubin

↓

Direct bilirubin > 15%

↓ (> 50%)

Conjugated Hyperbilirubinemia



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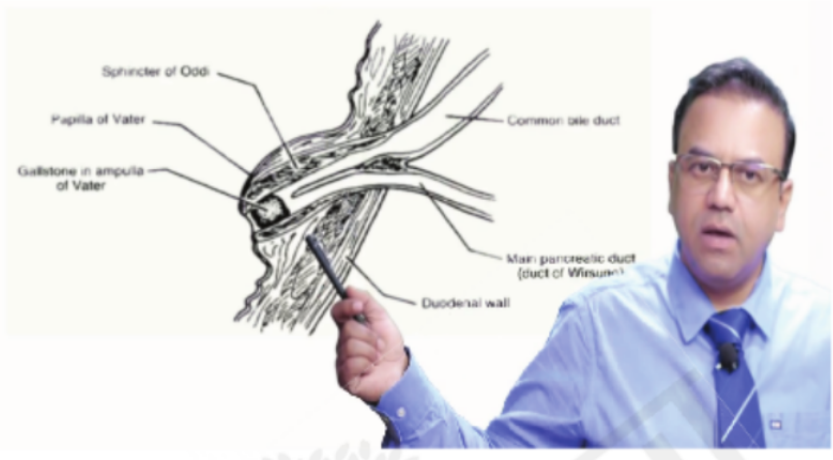

Jaundice

- ↑ Bilirubin
- Total Bilirubin (> 2.5 mg/dL)
- Yellowish discoloration in sclera & palate.

Prehepatic

- Hemolytic anemia
  ↑ unconj. bilirubin

Urine → Bilirubin ⊖
Urobilinogen ⊕.

DDx
- Gilbert Synd.
- Crigler Najar Sx.

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- ALT, AST → Normal.

Post Hepatic

Surgical causes
→ Gall stones.

Urine
Conjugated bilirubin ⊕.
Urobilinogen → ⊖.

USG
Bile duct dilatation

Hepatic

- Hepatitis
(eg: viral causes)

↑ unconjugated Bilirubin – dlt inflammation / injury in liver
↑ conjugated Bilirubin

(Whatever conjugation done, it has to undergo through various canaliculi to reach bile duct, because of the inflammation, canaliculi must be blocked, so conjugated bilirubin is not coming to intestine)

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Urine

Bilirubin +ve

uro bilinogen +ve

- ALT - ↑
- AST

Liver fibrosis

Ix

1) NAFLD - fibrosis score.
2) AST - Platelet ratio Index.
3) fibroscan (fibrotest)

Imaging

1) USG - fibroscan — VcTE (vibration controlled transient Elastography)
2) MR - Elastography

NAFLD - fibrosis Score

Parameters

- Age
- BMI

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- Diabetes
- AST
- ALT
- Platelets
- Albumin

APRI

$$\text{APRI} = \frac{\dfrac{\text{AST Level}}{\text{AST Upper Limit of Normal}}}{\text{Platelet Count } (10^9/\text{L})} \times 100$$

FibroSure Test

FibroSure (FibroTest)		ActiTest
Apolipoprotein A1	↓	
Haptoglobin	↓	
α_2-macroglobulin	↑	+ ALT ↑
Gamma glutamyltranspeptidase	↑	
Total bilirubin	↑	

High positive and negative predictive value for advanced fibrosis

Large indeterminate group

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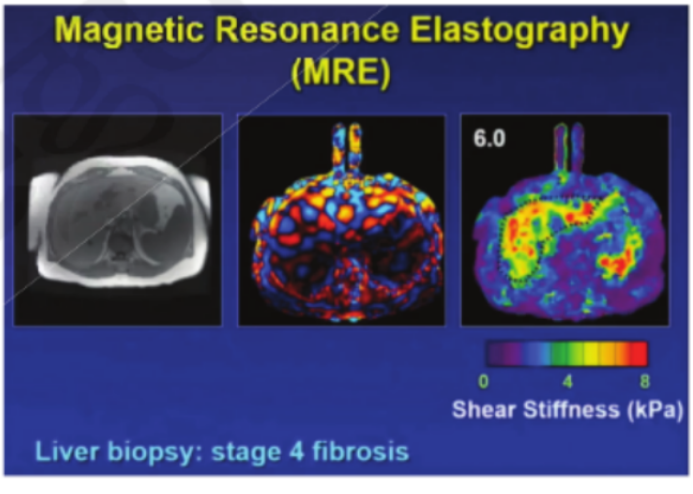
Elastography

MR- Elastography

Score >4 → Fibrosis

1·08:56·



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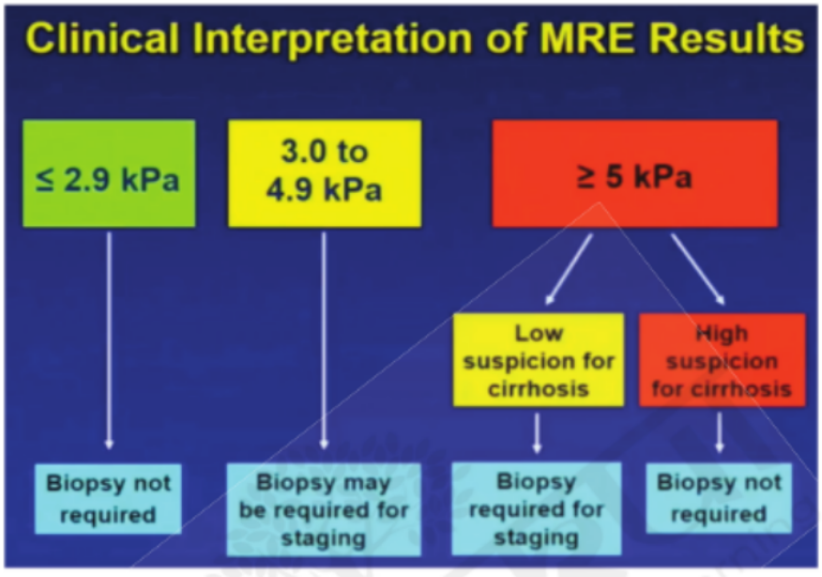

Chronic Hepatitis

- AST, ALT level → Moderate Range (b/w 200 - 400)
- Low Albumin
- R. Ratio >5.

ALT > Alk. Phos are.

Albumin – Half life – 28 days.

Viral Hepatitis → Hep B, Hep C

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Urine

Bilirubin +ve

uro bilinogen +ve

- ALT - ↑
- AST

Liver fibrosis

Ix

1) NAFLD - fibrosis score.
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Imaging

1) USG - fibroscan — VcTE (vibration controlled transient Elastography)
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NAFLD - fibrosis Score

Parameters

- Age
- BMI

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- Diabetes
- AST
- ALT
- Platelets
- Albumin

APRI

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FibroSure Test

FibroSure (FibroTest)		ActiTest
Apolipoprotein A1	↓	
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α_2-macroglobulin	↑	+ ALT ↑
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Total bilirubin	↑	

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MR- Elastography

Score >4 → Fibrosis

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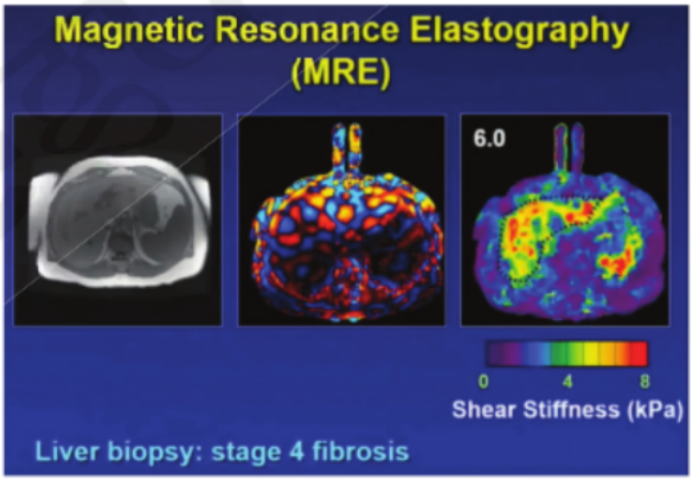

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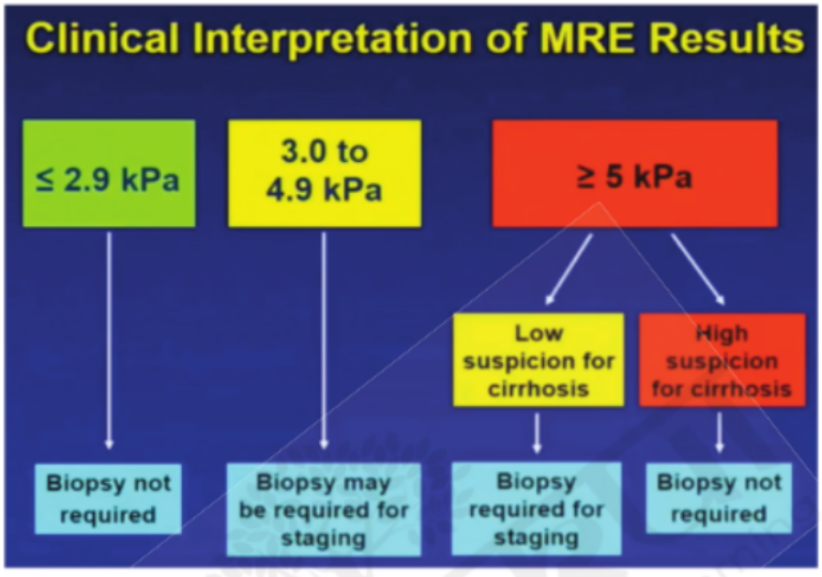

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ALT > Alk. Phos are.

Albumin – Half life – 28 days.

Viral Hepatitis → Hep B, Hep C

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- Hemochromatosis → Transferrin Saturation levels. (above 45%)
- AI hepatitis - SPEP
  ↳ polyclonal hyper-gammaglobulinemia
- Wilson's disease
- Alcoholic / NAFLD.

Markers
- Anti smooth muscle ab.
- LKM-1 Ab.

Note:

SPEP → Serum Protein Electrophoresis.

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HEPATITIS A & B – I

VIRAL HEPATITIS

A	B	C	D	E
Feco oral	Blood / Body fluids			Feco-oral
Acute	A/c (chronic)	A/c	A/c.	Acute (chronic in Immunocomp.)
IP → 30 days	60 days	50 days	60 days	40 days
Fulminant Hepatitis → (A → 0.2)	1%	0.1%	5-20%	1-2% (preg- 20%)

Least risk of Fulminant Hepatitis → C *

Incubation period

A – 30 days
E – 40 days
C – 50 days
B / D – 60 days.

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Acute Viral Hepatitis

- fatigue
- Jaundice
- Vomiting / Jaundice
- Nausea
- Arthralgia / Arthritis

LFT

ALT – ↑↑
ALP – ↑ } R > 5

Hepatitis A

- RNA virus
- Hepatovirus
- Picorna virus family

Spread

- close personal contact
- contaminated food & H_2O

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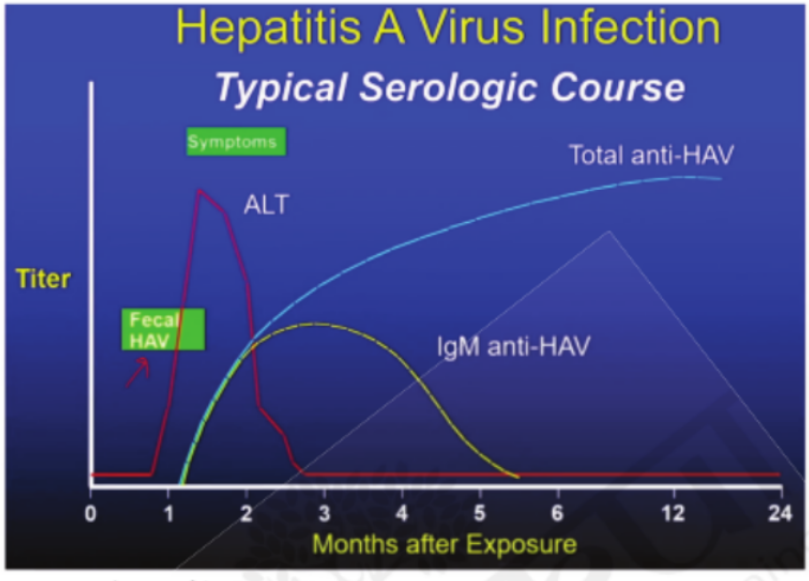

5 patterns

1) Anicteric

2) Symptomatic – Jaundice
Self limiting | 2 months.

3) Cholestatic – Jaundice
(> 10 weeks)

4) Relapsing – 2 or more bouts
of Acute – HAV → 6 – 10 wks.

5) Fulminant Hepatic Failure.

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Rx

- Supportive
- Hydration
- Diarrhoea - I/v fluids

Vaccines

- HAVRIX
- VAQTA
- TWINRIX

2 doses given - 1st - 0
2nd - 6-12 months.

TABLE 78-2 Recommended Regimens for Hepatitis A Vaccination*

Vaccine	Schedule	Age (yr)	Dose	Volume (mL)	Dosing Schedule
HAVRIX	Standard	1-18	720 ELU	0.5	0, 6-12 months
	Standard	>18	1440 ELU	1	0, 6-12 months
	Accelerated	≥1	Single age-appropriate dose	Age appropriate	≥2 weeks prior to travel†
	Postexposure prophylaxis	≥1	Single age-appropriate dose	Age appropriate	<2 weeks after exposure‡
VAQTA	Standard	1-18	25 U	0.5	0, 6-18 months
	Standard	>18	50 U	1	0, 6-18 months
	Accelerated	≥1	Single age-appropriate dose	Age appropriate	≥2 weeks prior to travel†
	Postexposure prophylaxis‡	≥1	Single age-appropriate dose	Age appropriate	<2 weeks after exposure‡
TWINRIX	Standard	≥18	720 ELU HAV, 20 mg HBV	1	0, 1, 6 months
	Accelerated	≥18	720 ELU HAV, 20 mg HBV	1	0, 7, 21-30 d

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Monovalent Hepatitis A Vaccine, Inactivated

- Universal: all children 1-2yo
- Recommended: travel to endemic areas (long notice), risk groups, CLD

	Dose	Schedule
Havrix®[1]	1440 EL.U. in 1.0 mL IM	0, 6 to 12 months
Vaqta®[2]	~50 U in 1 mL IM	0, 6 to 12 months

Pre-Exposure Prophylaxis

- Hep. A Immunoglobulin

Visting Endemic Area. (< 2 weeks)

Post Exposure

- Within 14 days.
- HA Ig.

Hepatitis B

- Hepedna — 1
- DNA vaccine.

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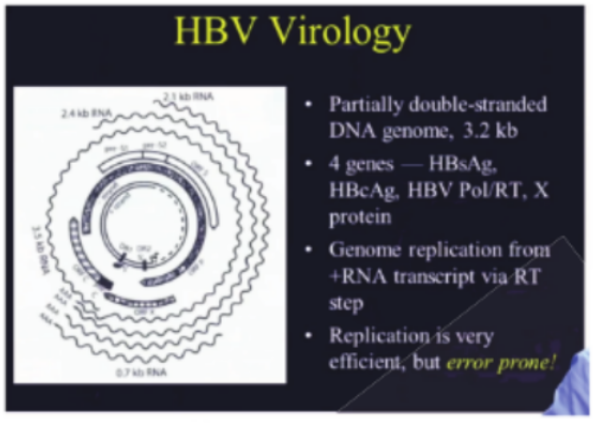

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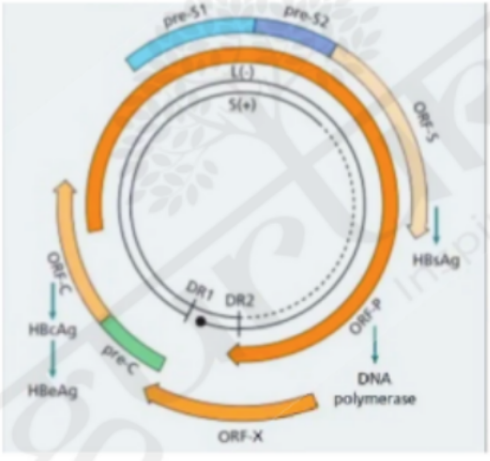

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S, Pre-S2, Pre-S1
↓ ↓ ↓
HbsAg small Medium Large

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Core Ag → anti HbcAg · Ab.

Pre core mutant → No ability to produce HbeAg.
High risk of developing HCC & cirrhosis.

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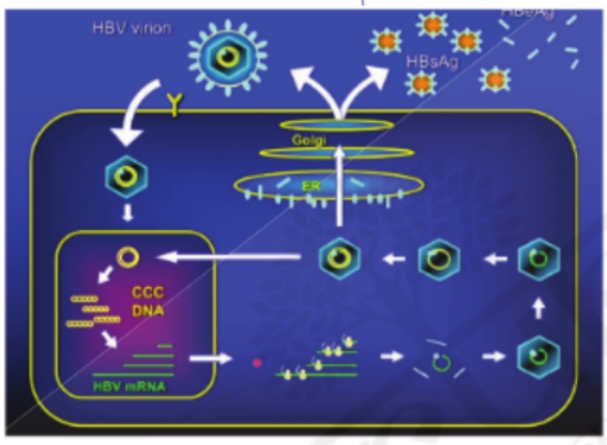

Mode of Transmission

- Vertical
  - Mother → Baby (chronic)
- Horizontal
  - Trans-sexual (M/C)
  - Needle stick
  - Blood transfusion
  - Injecting Drug Use
  - Home hold contacts

95% chronic

5% → chronic

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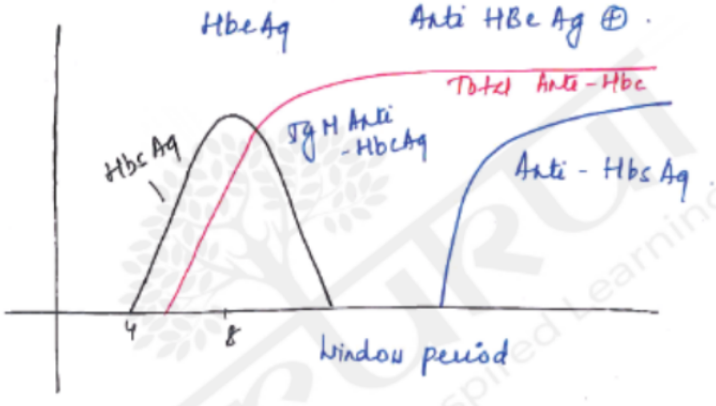
Serology

Acute Hepatitis B.

Symptoms.

HBV DNA ↑



After 4 weeks of infection → surface Ag comes out of blood (1st Marker)

After 1-2 weeks – IgM anti-HbcAg produces (1st Antibody)

- Hbe Ag ⊕
- HBV DNA level ↑

Symptoms ⊕ – later

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Symptoms manifest around 8 weeks.

Markers of viral Replication

- HBV-DNA → Quantitative
- HbeAg → Qualitative

Non-Replicative Phase

Anti-HBe Ag.

Window period

No HBsAg & No antibodies against surface Ag.
(Low window period)

Main markers present → IgM Anti HbcAg
± anti HbeAg.

Acute HBV

- 95% pts → Recover
- Supportive tx.
- Mostly resolve

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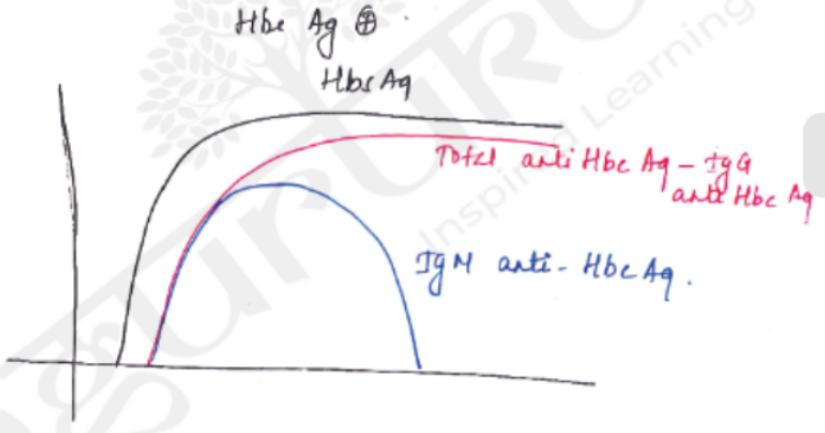
Extrahepatic Manifestations

- Polyarthritis Nodosa.
- Cryoglobinemia
- Skin rash → Gianotti Crosti Sx.

Chronic Hepatitis B.

Serology



Markers present:

HbsAg & IgG anti HbcAg.

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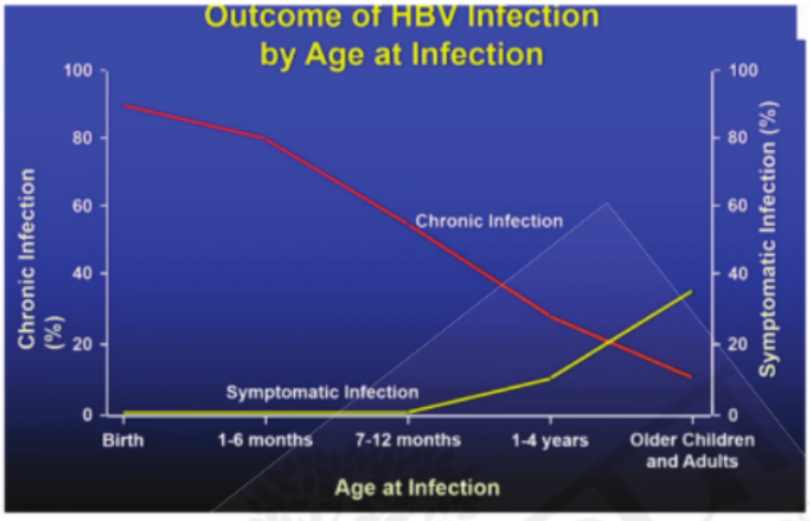

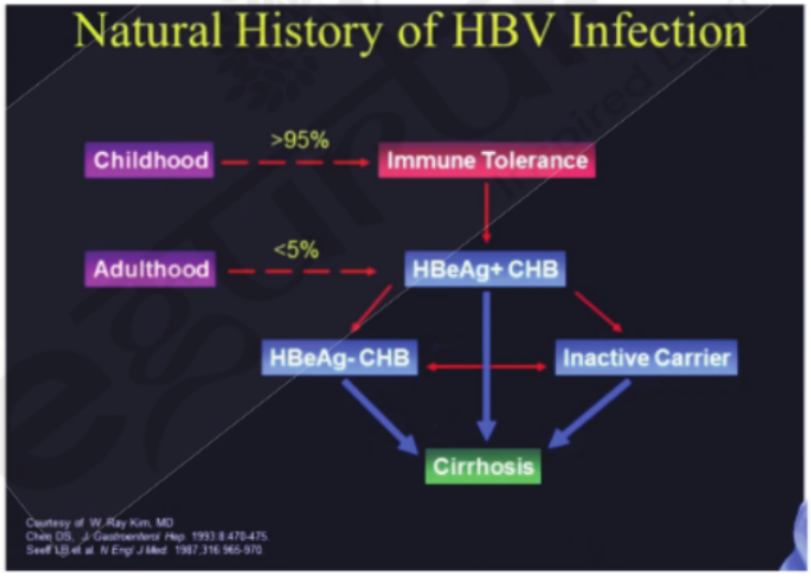

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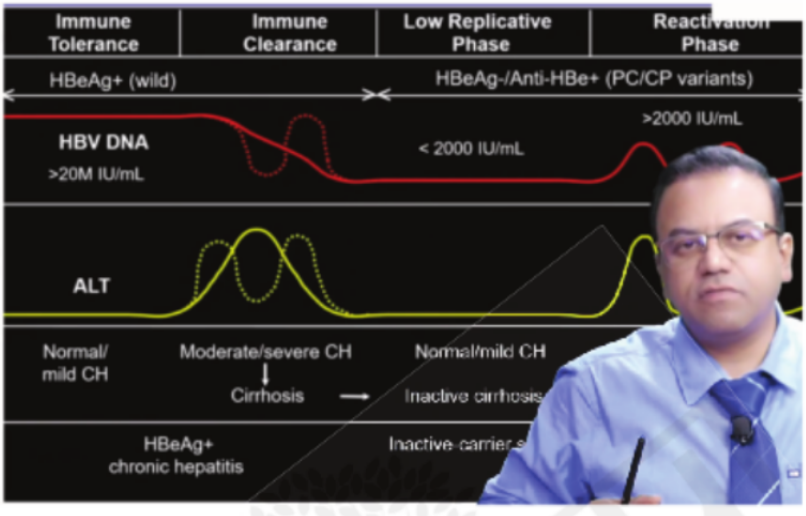

TABLE 6. Diagnostic Criteria and Definitions for Chronic Hepatitis B

Chronic Hepatitis B (CHB)

1. HBsAg present for ≥6 months
2. Serum HBV DNA varies from undetectable to several billion IU/mL
3. Subdivided into HBeAg positive and negative. HBV-DNA levels are typically >20,000 IU/mL in HBeAg-positive CHB, and lower values (2,000-20,000 IU/mL) are often seen in HBeAg-negative CHB.
3. Normal or elevated ALT and/or AST levels
4. Liver biopsy results show chronic hepatitis with variable necroinflammation and/or fibrosis

Immune-Tolerant CHB

1. HBsAg present for ≥6 months
2. HBeAg positive
3. HBV-DNA levels are very high (typically >1 million IU/mL).
4. Normal or minimally elevated ALT and/or AST
4. Liver biopsy or noninvasive test results showing no fibrosis and minimal inflammation

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Immune-Active CHB

1. HBsAg present for ≥6 months
2. Serum HBV DNA >20,000 IU/mL in HBeAg-positive CHB and >2,000 IU/mL in HBeAg-negative CHB
3. Intermittently or persistently elevated ALT and/or AST levels
4. Liver biopsy or noninvasive test results show chronic hepatitis with moderate or severe necroinflammation and with or without fibrosis

Inactive CHB

1. HBsAg present for ≥6 months
2. HBeAg negative, anti-HBe positive
3. Serum HBV DNA <2,000 IU/mL
4. Persistently normal ALT and/or AST levels
5. Liver biopsy confirms absence of significant necroinflammation. Biopsy or noninvasive testing show variable levels of fibrosis

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HEPATITIS B - II

Anti HbsAg +ve → Vaccinated

HbsAg +
IgM Anti HbcAg → Acute Hepatitis B

Anti HbsAg
IgG Anti HbcAg → Recovered

HbsAg +
IgG anti HbcAg + → chronic Hep. B

No HbsAg
No anti HbsAg → Window period
IgG anti HbcAg → Remote, Recovered
False positive +

HBsAg	ANTI-HBs	ANTI-HBc	HBeAg	ANTI-HBe	INTERPRETATION
+	–	IgM	+	–	Acute hepatitis B, high infectivity[a]
+	–	IgG	+	–	Chronic hepatitis B, high infectivity
+	–	IgG	–	+	1. Late acute or chronic hepatitis B, low infectivity 2. HBeAg-negative ("precore-mutant") hepatitis B (chronic or, rarely, acute)
+	+	+	+/–	+/–	1. HBsAg of one subtype and heterotypic anti-HBs (common) 2. Process of seroconversion from HBsAg to anti-HBs (rare)
–	–	IgM	+/–	+/–	1. Acute hepatitis B[b] 2. Anti-HBc "window"
–	–	IgG	–	+/–	1. Low-level hepatitis B carrier 2. Hepatitis B in remote past
–	+	IgG	–	+/–	Recovery from hepatitis B
–	+	–	–	–	1. Immunization with HBsAg (after vaccination) 2. Hepatitis B in the remote past 3. False-positive

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HBsAg	ANTI-HBs	ANTI-HBc	HBeAg	ANTI-HBe	INTERPRETATION
+	–	IgM	+	–	Acute hepatitis B, high infectivity[a]
+	–	IgG	+	–	Chronic hepatitis B, high infectivity
+	–	IgG	–	+	1. Late acute or chronic hepatitis B, low infectivity 2. HBeAg-negative ("precore-mutant") hepatitis B (chronic or, rarely, acute)
+	+	+	+/–	+/–	1. HBsAg of one subtype and heterotypic anti-HBs (common) 2. Process of seroconversion from HBsAg to anti-HBs (rare)
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–	+	IgG	–	+/–	Recovery from hepatitis B
–	+	–	–	–	1. Immunization with HBsAg (after vaccination) 2. Hepatitis B in the remote pa 3. False-positive

Test	Acute Hepatitis B	Past Exposures (Immunity)	Vaccinated	Chronic eAg(+) (replicative)	Chron eAg(-) (replicative)	Carrier
HBsAg	+	-	-	+	+	+
Anti-HBs	-	+	+	-	-	-
HBeAg	+	-	-	+	-	-
anti-HBe	-	+/-	-	-	+	+
anti-HBc	+	+	-	+	+	+
IgM anti-HBc	+	-	-	-	-	
HBV DNA (PCR)	+	-	-	>20,000 IU/mL	>2,000 IU/mL	
ALT	Elevated	Normal	Normal	Elevated	Elevated	

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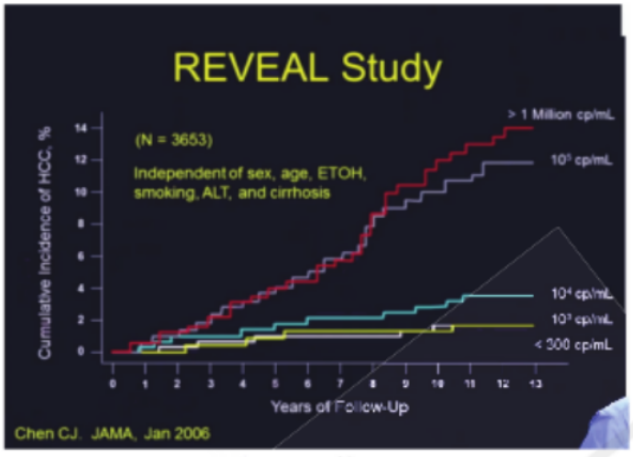

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Extrahepatic Manifestations

1) PAN
2) Membranous GN
3) EMC
4) papular acrodermatitis → Gianotti Crosti Sx.

Chronic Hep. B

HbsAg + IgG anti HbcAg +

HbeAg +

HBV DNA <20,000
↓
ALT - (N)
↓
Follow up
No Rx

HBV DNA >20,000
↓
ALT
ALT - (N)
ALT ↑ (<2 xULN) - Rx.
x2 upper limit (N)

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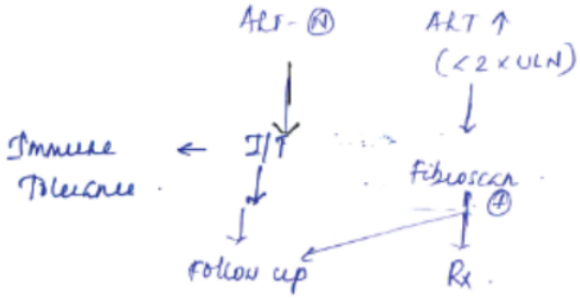
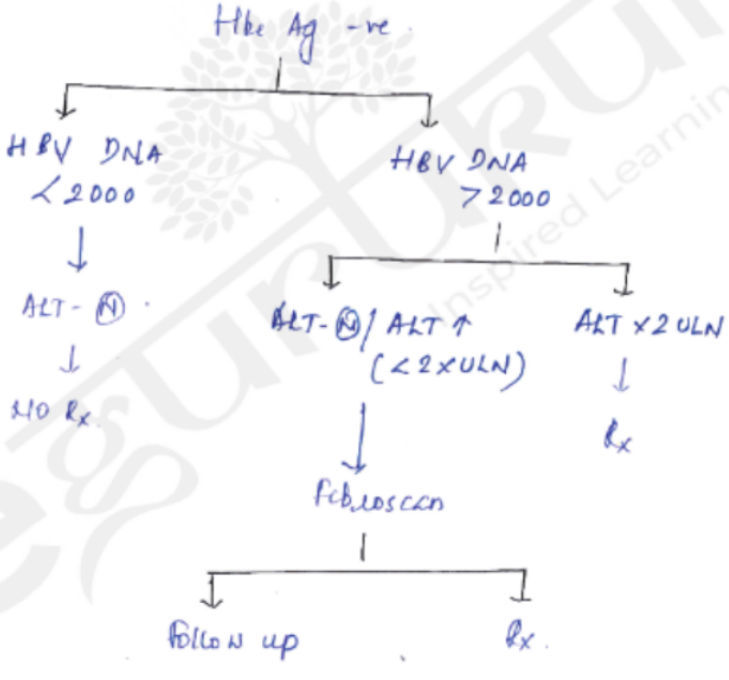


Chronic Hep B



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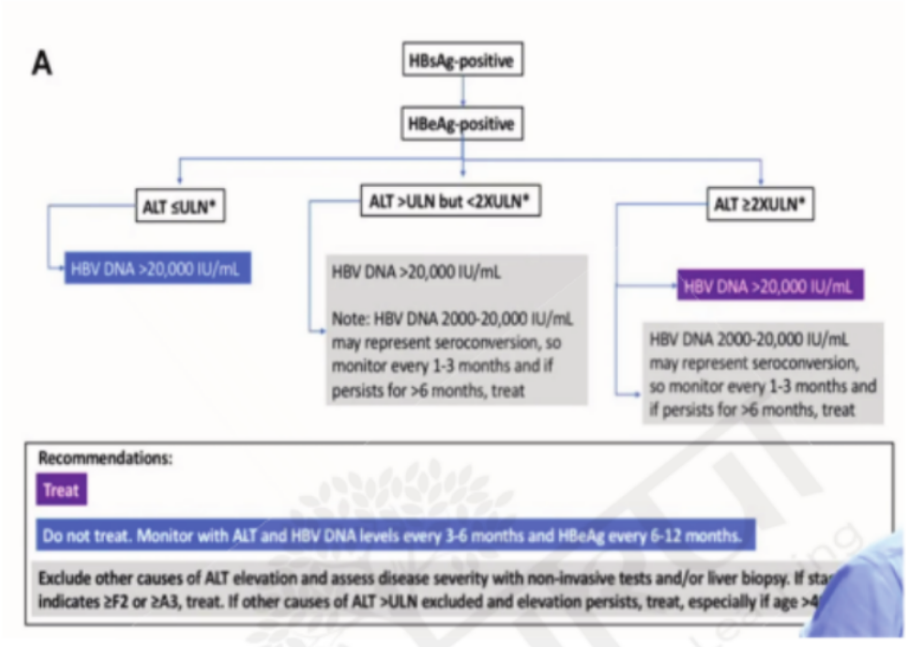

Management

Peg INF α	Nucleoside/Nucleotide
- Inj → S/c Inj.	→ Oral
- Poorly tolerated	- well tolerated
- No resistance	- Resistance int
	Tenofovir - resistance absent
- finite Rx 48 wks Rx should be stopped.	- Given for longer duration

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Contraindications of IFNα.

- Immunocompromised patients
- Cirrhosis (Decompensated)
- Pregnancy
- Post transplant
- Psychiatric disease.

- Desire for finite therapy (see below).
- Anticipated tolerability of treatment side effects.
- Comorbidities: peg-IFN is contraindicated in persons with autoimmune disease, uncontrolled psychiatric disease, cytopenia, severe cardiac disease, uncontrolled seizures, and decompensated cirrhosis.
- Previous history of lamivudine resistance (entecavir is not preferred in this setting).
- Family planning: finite therapy with peg-I

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Nucleotide / Nucleoside Analogues.

→ Tenofovir Alafenamide (Prodrug of Tenofovir)

→ Tenofovir

→ Entecavir

→ Adefovir

→ Telbovudine

→ Lamivudine

Combined HIV & HBV.

- Emtricitabine
- Tenofovir
- Lamivudine – dev. resistance in HBV rapidly.

44·35

Clinical Situation	First-Line Therapy
Treatment naïve	Entecavir or tenofovir
Prior lamivudine or telbivudine exposure	Switch to tenofovir
Proved lamivudine resistance	Switch to tenofovir
Proved adefovir resistance	Switch to entecavir
Primary drug failure with both lamivudine and adefovir*	Switch to tenofovir or entecavir
Proved entecavir resistance	Switch to tenofovir
Proved telbivudine resistance	Switch to tenofovir
Persistent low-level viremia during treatment with high-genetic-barrier drug	Continue tenofovir or entecavir or switch to the other
Treatment naïve, decrease in GFR (60-90 mL/min)	Entecavir

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Pt is HBe Ag +.

Rx → till seroconversion → Stop.
(Nucleoside Analogues) to anti HBe Ag 12 months.

↓

HBe Ag -ve → life long.

Vaccine

RECOMBIVAX-HB (Merck)[a]			
Infants, children (<1–10 years)	3	5 µg (0.5 mL)	0, 1–2, 4–6
Adolescents (11–19 years)	3 or 4	5 µg (0.5 mL)	0–2, 1–4, 4–6 or 0, 12, 24 o
	Or		
Adults (≥20 years)	2	10 µg (1 mL)	0, 4–6 (age 11–15)
Hemodialysis patients[b]	3	10 µg (1 mL)	0–2, 1–4, 4–6
<20 years	3	5 µg (0.5 mL)	0, 1, 6
≥20 years	3	40 µg (4 mL)	0, 1, 6
ENGERIX-B (GlaxoSmithKline)[c]			
Infants, children (<1–10 years)	3 or 4	10 µg (0.5 mL)	0, 1–2, 4–6 or 0, 1,
Adolescents (10–19 years)	3 or 4	10 µg (0.5 mL)	0, 1–2, 4–6 or 0, 1
Adults (≥20 years)	3 or 4	20 µg (1 mL)	0–2, 1–4, 4–6 or 0,
Hemodialysis patients[b]			
<20 years	4	10 µg (0.5 mL)	0, 1, 2,
≥20 years	4	40 µg (2 mL)	0, 1,

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Definition of HBV cure

Functional cure (equivalent to resolved acute infection).

Sustained / durable HBsAg loss ± or ± out anti HBs seroconversion, ± undetectable serum DNA, but persistence of cccDNA which is not transcriptionally active, allowing treatment cessation.

Complete cure

as for functional cure, but ± cccDNA clearance.

Generation of the cccDNA nuclear reservoir

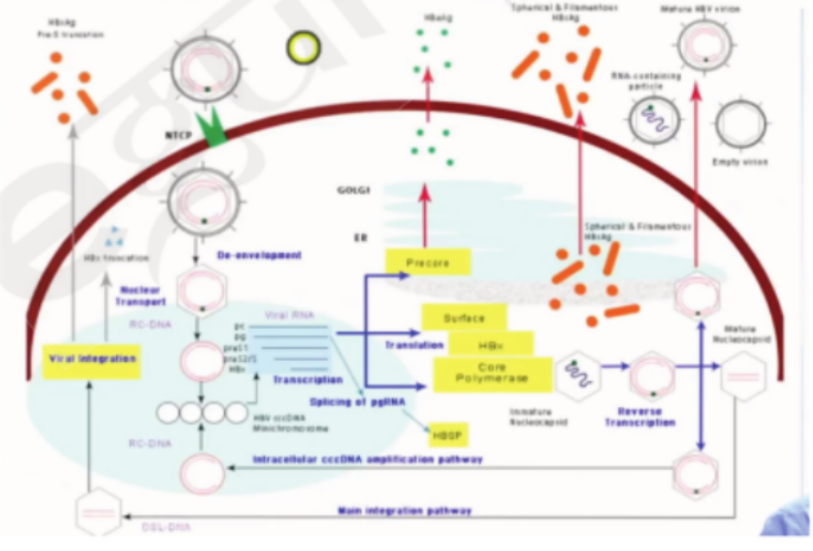

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TABLE 79-7 Recommended Postexposure Prophylaxis for HBV According to the Vaccination Status of the Exposed Person

51.06

Vaccination Status of Exposed Person	Recommended Prophylaxis
Unvaccinated	HBIG (0.06 mL/kg) and initiate the hepatitis B vaccine series
Previously vaccinated:	
Known responder*	None
Known nonresponder	HBIG × 2 doses (1 month apart) OR HBIG × 1 dose and initiate revaccination
Antibody response unknown	Test for anti-HBs If adequate*: No treatment If inadequate†: HBIG × 1 dose and give vaccine booster dose

Risk of infection in unvaccinated person → 3-30%

0.8 – 8% → HIV pts.

- Risk of HCC - Depends on viral load
- ↓
  - Males at high risk
  - Family H/o.
  - Fibrosis ↑
  - HBV - DNA ↑

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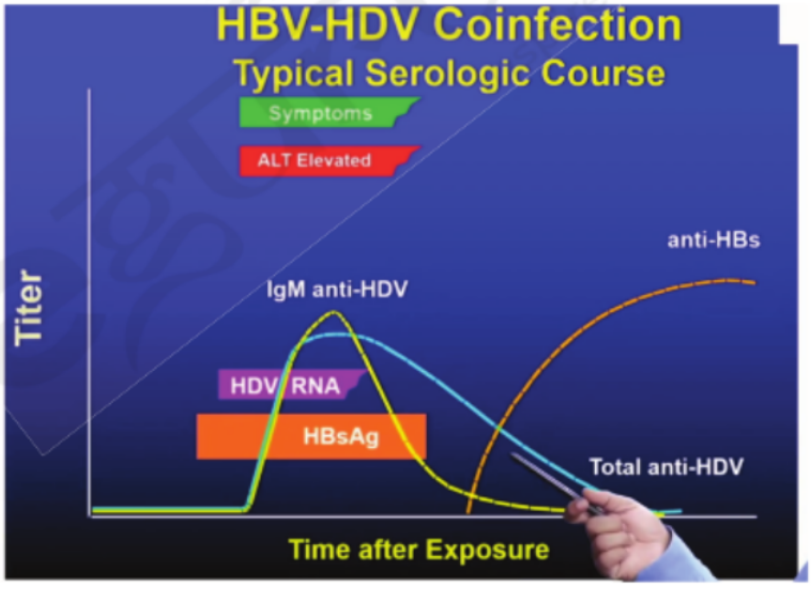
Hepatitis D

- Delta virus
- Coinfection - C HBV + HDV → } ↑ Risk of fulminant hepatitis
  Acute + HDV
- Super infection
  - IgM anti HBcAg
  - HBsAg + Anti-HDV

- IgG anti HBcAg + Anti HDV } - progression to cirrhosis / HCC
  HBsAg



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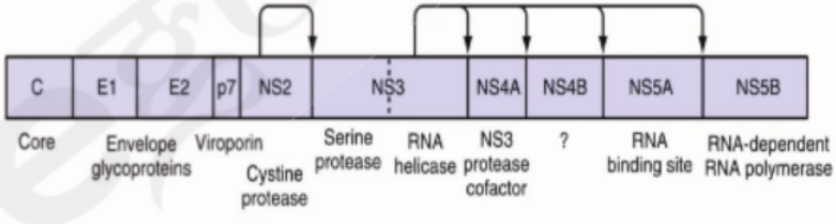
HEPATITIS C & E

Hepatitis C

- Hepacivirus
- Flavivirus - subtype
- RNA virus.

Transmission

- Injection drug use
- Blood transfusion
- Needle stick injuries
- Tattooing
- Sexual



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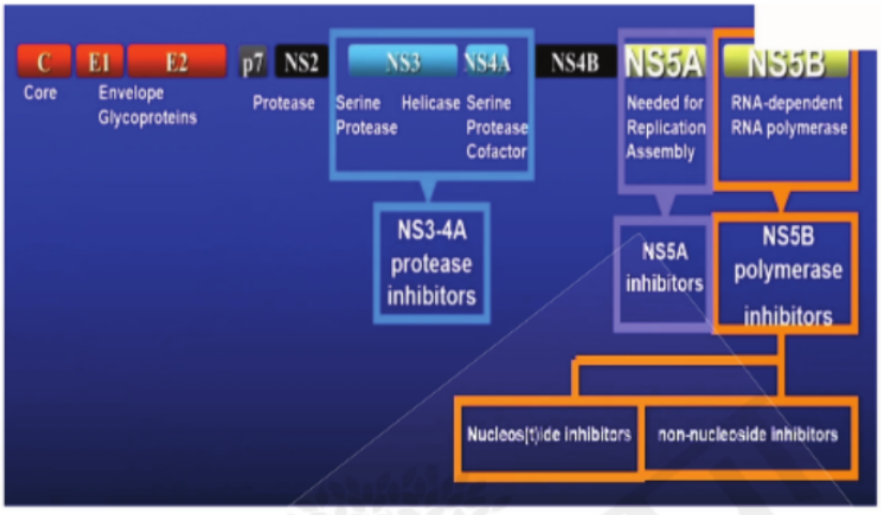

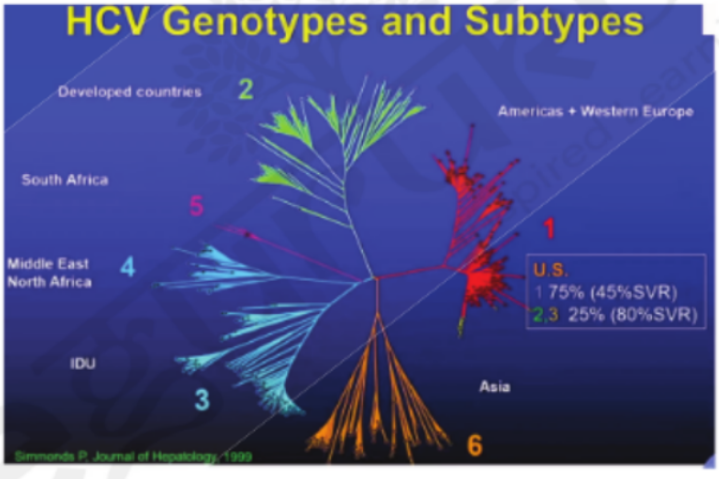

India → Genotype 3.

US → Genotype 1

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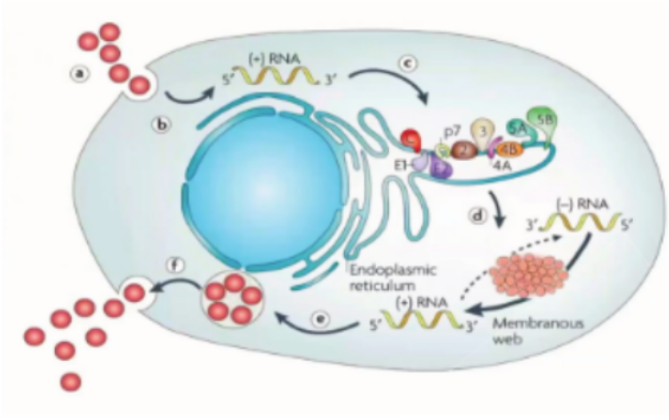
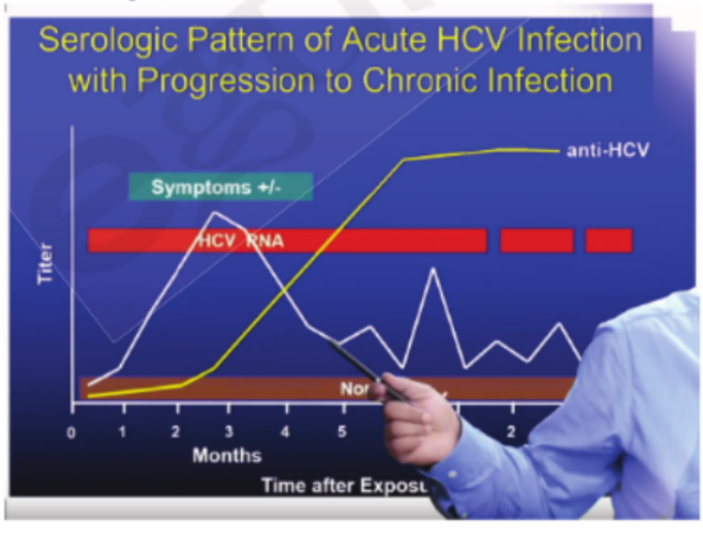


5.52

Incubation period – 50 days.
When virus starts replicating
↓
- HCV RNA ↑
- Anti HCV ↑

Symptoms – +/-



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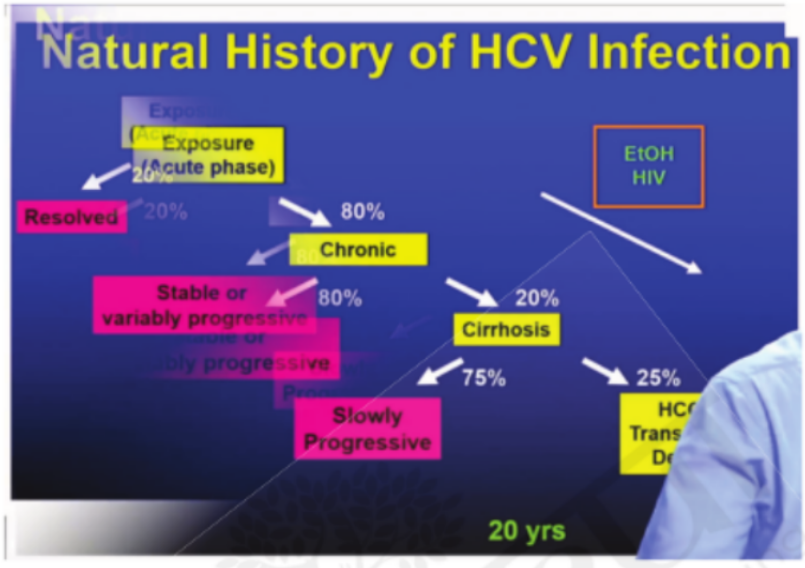

Extra Hepatic Manifestations

- Lichen planus.
- Porphyria cutanea tarda. (PCT)
- Mixed cryoglobulinemia
- Non Hodgkins lymphoma.
- AI thyroiditis.
- Monoclonal gammopathies
- Membranoproliferative GN. (MPGN).

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Associations of Hep C. (Not proven)

- DM
- polyarthritis
- Thyroid cancer
- RCC
- Vitiligo
- Idiopathic pulmonary fibrosis

16.50

Investigations

- LFT
- Anti HCV
- HCV - RNA

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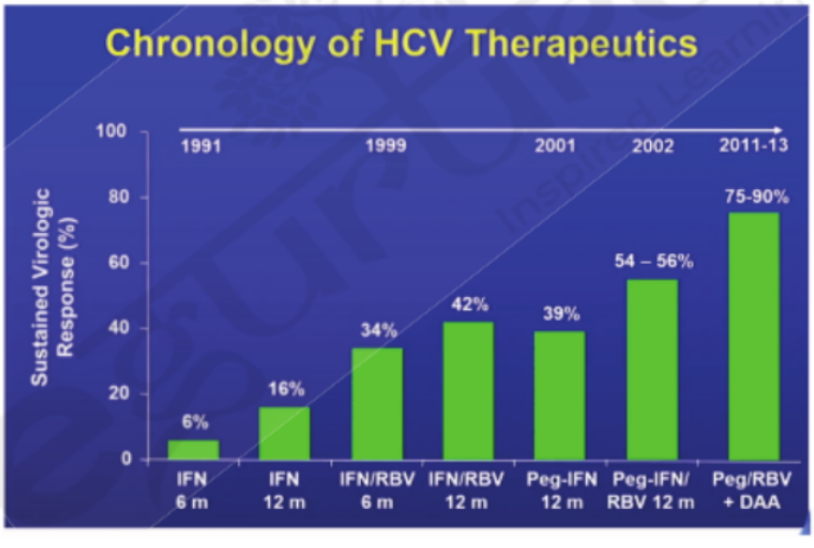
- Liver fibrosis
  - ↳ fibrosure
  - ↳ AST - platelet ratio Index (APRI)
  - ↳ FIB - 4
  - → fibroscan
  - → MRE
  - → Liver biopsy.
- RFT



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NS3/4A Protease Inhibitors		
Glecaprevir	1–6	300 mg orally once daily
Grazoprevir	1 and 4	100 mg orally once daily
Paritaprevir	1 and 4	150 mg orally once daily
Simeprevir	1 and 4	150 mg orally once daily
Voxilaprevir	1–6	100 mg orally once daily
NS5A Inhibitors		
Daclatasvir[a]	1–6	60 mg orally once daily
Elbasvir	1–6	50 mg orally once daily
Ledipasvir	1, 4–6	90 mg orally once daily
Ombitasvir	1 and 4	25 mg orally once daily
Pibrentasvir	1–6	120 mg orally once daily
Velpatasvir	1–6	100 mg orally once daily

NS5B Nucleos(t)ide Polymerase Inhibitor		
Sofosbuvir	1–6	400 mg orally once daily
NS5B Non-Nucleos(t)ide Polymerase Inhibitor		
Dasabuvir	1 and 4	250 mg orally twice daily

27.37

DAA's for HCV

- NS3A Protease inhib
  - Simeprevir
  - Paritaprevir
  - Grazoprevir
  - Glecaprevir
  - Voxilaprevir
- NS5B Polymerase inhib
  - Sofosbuvir (Nuc)
  - Dasabuvir (non-Nuc)
- NS5A inhib
  - Ledipasvir
  - Ombitasvir
  - Elbasavir
  - Pibrentasvir
  - Velpatasvir
  - Daclatasvir

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Characteristic	NS3 Protease inhibitors	Nucleos(t)ide NS5B Polymerase inhibitors	Nonnucleoside NS5B Polymerase inhibitors	[illegible]
Potency	High; Variable among HCV genotypes	Moderate-high; **Pangenotypic**	Variable; Variable among HCV genotypes	High; increasingly pangenotypic
Barrier to Resistance	Low	**High**	Very Low	Low
Drug Interaction Potential	High	**Minimal**	Variable	Low to moderate

H/I combination → Sofosbuvir + Velpatasvir. (12 weeks)

Characteristic	NS3 Protease inhibitors	Nucleos(t)ide NS5B Polymerase inhibitors	Nonnucleoside NS5B Polymerase inhibitors	[illegible]
Potency	High; Variable among HCV genotypes	Moderate-high; **Pangenotypic**	Variable; Variable among HCV genotypes	High; increasingly pangenotypic
Barrier to Resistance	Low	**High**	Very Low	Low
Drug Interaction Potential	High	**Minimal**	Variable	Low to moderate

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cirrhosis / past treatment
↓
Glecaprevir + Pibrentasvir } 16 weeks.

Past DAA experienced (Direct Acting Agents)
- Sofosbuvir
- Velpatasvir
- Voxilaprevir
} 12 weeks.

Renal Dysfunction
Elbasvir + Grazoprevir } 12 weeks.

DAA Prior Exposure	Approved Therapies	
NS3/4A alone		
NS5A alone	Gle/ Pib X 16 weeks	Sof/ Vel/ Vox X 12 weeks
NS5A + NS3/4A		Sof/ Vel/ Vox X 12 weeks

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Category of patients to screen for HCC

- Asian Men > 40 yrs.
  Women > 50 yrs.
- African > 20 yrs.
- F/H + → HCC.
- > 40 yrs. ↑ ALT, High viral load

HCV - ↑ INF → ↓ HBV
↓
Viral load → ↑ Reaction - HBV.
Decreases

Hepatitis E

- RNA virus
- Hepevirdae family
- M/c cause of Acute Hepatitis in India.
- Feco-oral spread
- No parenteral / sexual transmission
  Vertical transmission possible.

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Symptoms

- Jaundice
- Fatigue
- Right upper quadrant pain
- Nausea / vomiting
- ALT ↑
- Anicteric
- No vaccine available
- Pregnancy (3rd trimester) → Mortality ↑ (25%)

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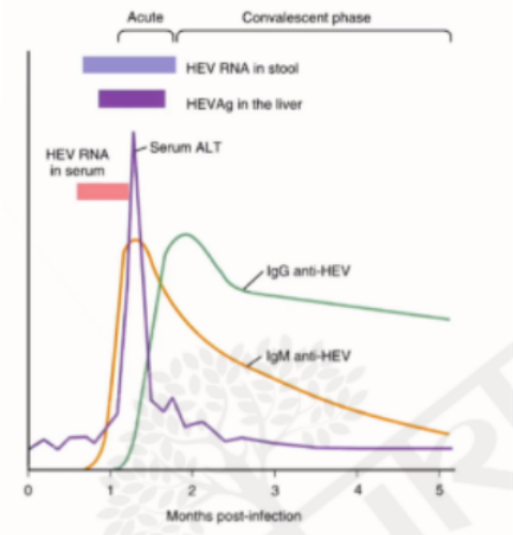
- Transplacental transmission ⊕

44·35



Rx

- Supportive
- Immunocompromised → post liver transplant
  ↓
  → chronic phase
  ↓
  Rx — Oral Ribavirin

Dx

Anti - HEV - IgM → Acute Hep E
  ↳ IgG — Recovered from Hep E

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Hepes simplex HEV

- ALT ↑ > 5000
- No jaundice.

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NAFLD & NASH

NAFLD
(Non - Alcoholic Fatty liver disease)

NAFLD
↓
NASH - Non Alcoholic Steato Hepatitis
↓
Cirrhosis
- Decompensated (Transplant)
- HCC

Risk factors

- Dyslipidemia
- HTN
- Obesity
- DM

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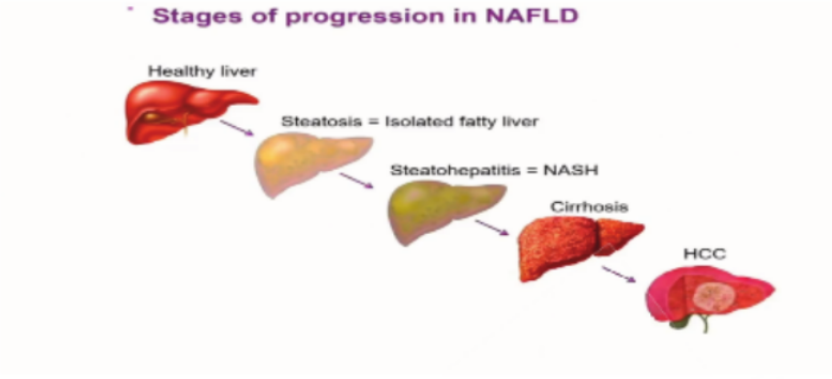

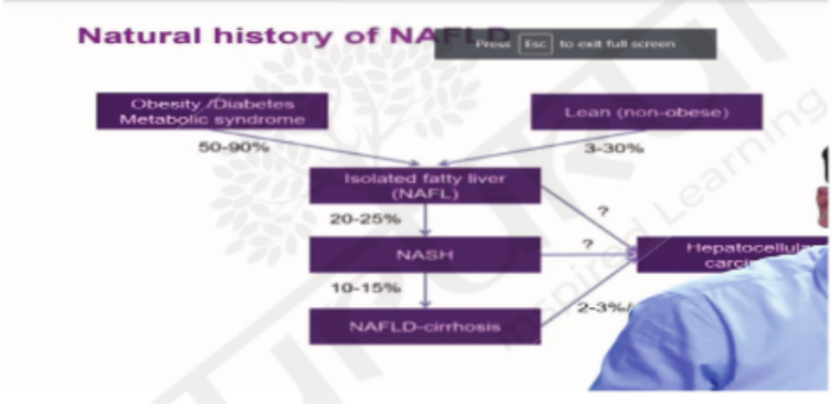

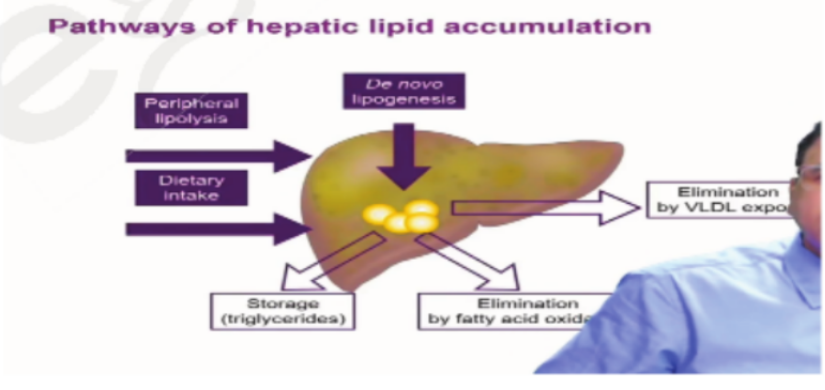

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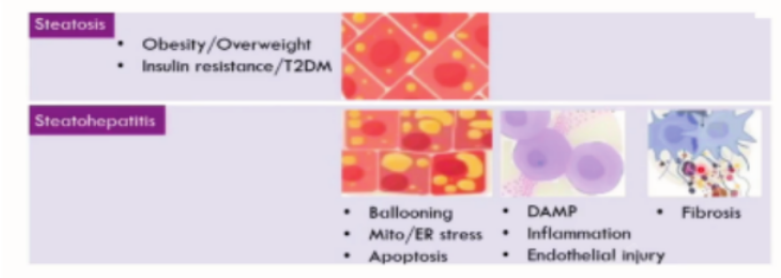

6.59
8.80

Drew, Nature 550: S102 (2017)

Steatosis
↓
Steatohepatitis → Altered adipokines.
→ Gut → Microbiota dysbiosis.
→ DAMP – Damage associated Molecular pattern
Trigger Inflammation
↓
Fibrosis.

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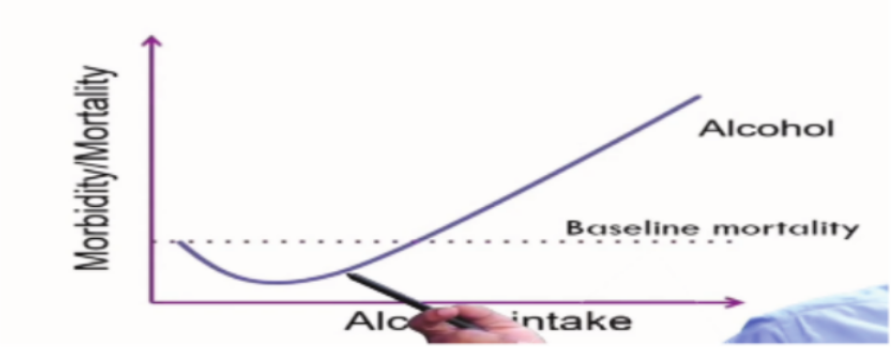

History

- Men > 3 drinks / day.
- Females > 2 drinks / day

Background

- Obesity / Dyslipidemia
- HTN / DM.

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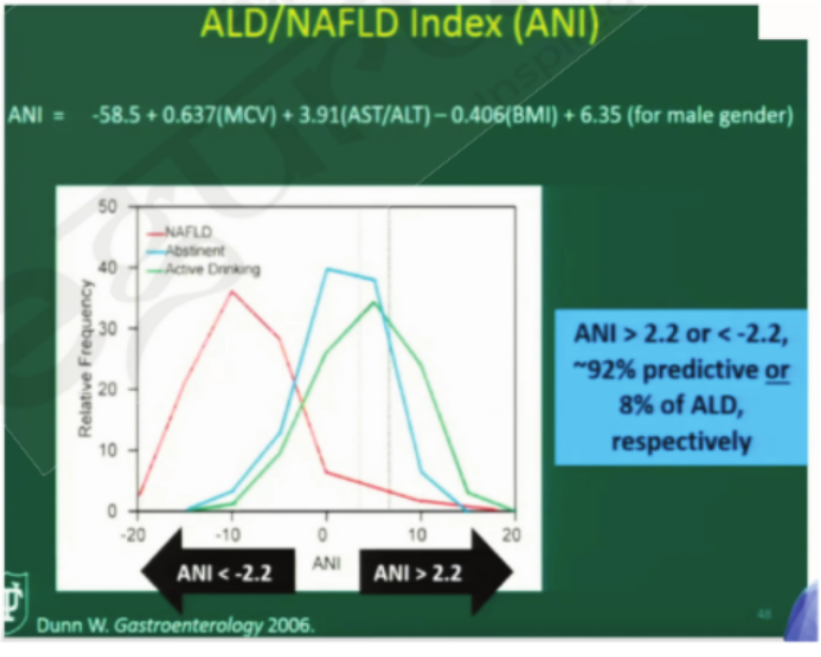
AST : ALT > 2 → Alcoholic liver Dz. (ALD)

AST : ALT < 1 → NAFLD / NASH.

AST : ALT = 1 or > 1 → Cirrhosis (AST > ALT)

ALD / NAFLD Index (ANI)

ANI > 2.2 or < -2.2



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	NASH	ASH
Alcohol use	+/-	+++
Glucose Intolerance	++	+/-
Obesity	++	+/-
* HDL	Low	Increased
AST : ALT	<1	>2
* MCV	+	+++
Jaundice	+	++
* GGT	+	>400
* ANI	<2.2	>2.2

Estimation of liver fat content by imaging

	Ultrasound	CT	MR	CAP
Sensitivity	++	++	+++	++
Specificity	+	+++	++++	++
Cost	+	+++	++++	+

- U/S is cost-effective, not quantitative, limited value in mild steatosis (may miss it if liver fat content <30%)
- CT may quantify fat by liver/spleen attenuation ratio, becomes reasonably accurate if liver fat content >30%
- MR is very accurate, either as proton spectroscopy (MRS) or chemical shift-based fat fraction (PDFF) measurements
- FibroScan CAP (controlled attenuation parameter) calculated by fat-induced distortion of liver stiffness measurements

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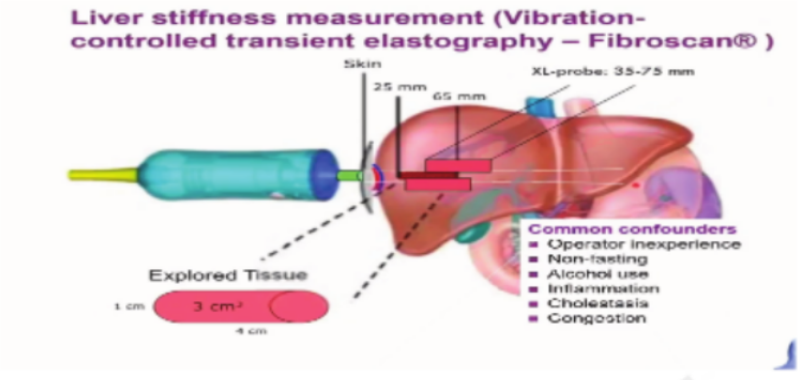

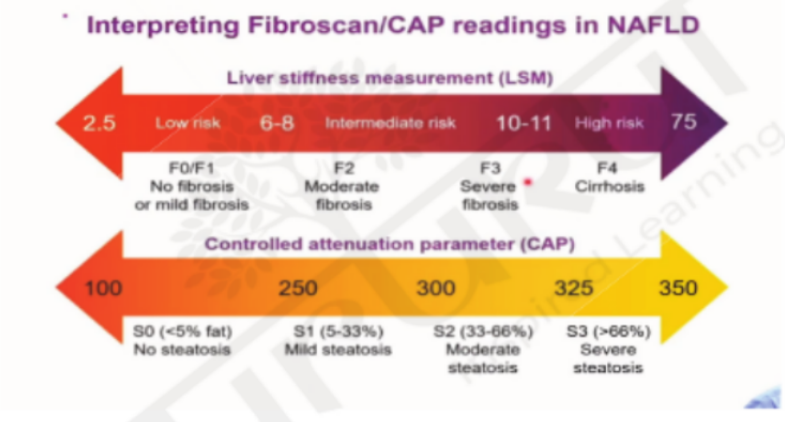

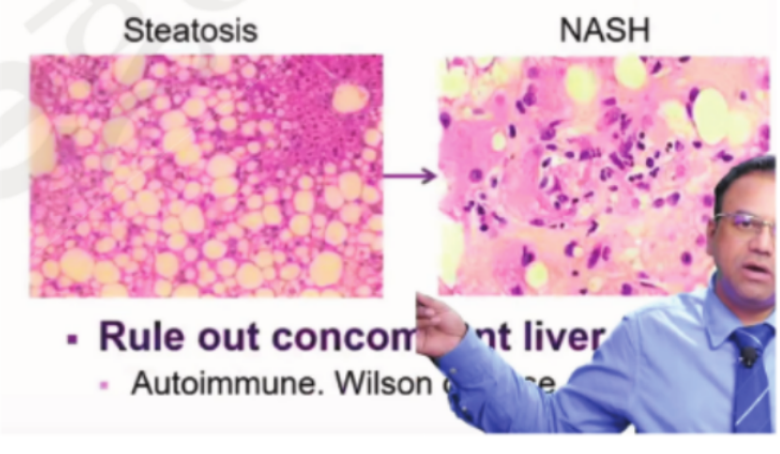

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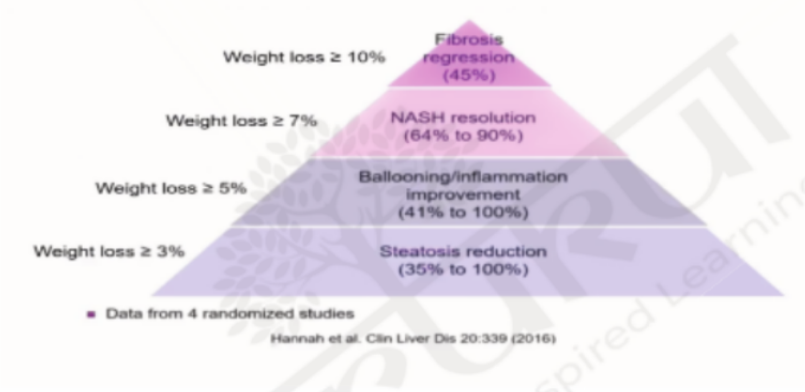
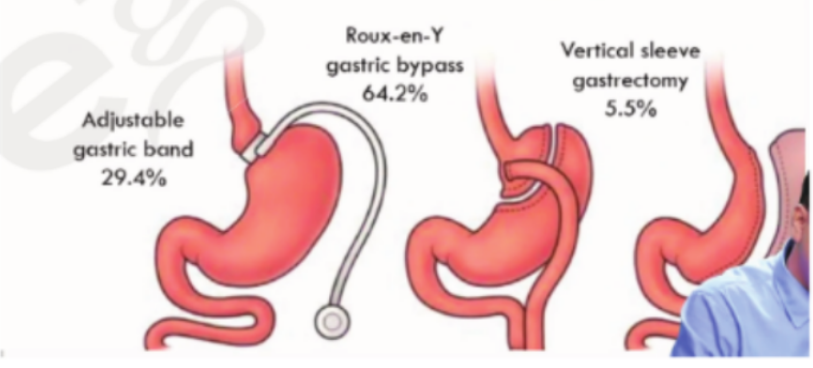
Rx → NAFLD | NASH

- Diet
- Physical Activity
- Pharmacotherapy



)15) ospective cohort e, France	52 weeks follow-up	NASH resolvec Fibrosis improved ≥1 in 34% and in 47% of those with F2



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Pharmacotherapy

1) Thiazolidione – (TZD) – Pioglitazone
2) GLP-1 Analogs.
3) Vit E
4) Obeticholic acid
5) Apoptosis Inhibitor – Selonsertib

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ALCOHOLIC LIVER DISEASE

- Males > 3 drinks / day
- Females > 2 drinks / day

1 drink = 10 gm of alcohol
(India) = 30 ml of spirit

Binge drinking

Drinking alcohol which increases blood alcohol level to 80 mg/dL or in 2 hrs.

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Binge drinking – 3 drinks in female
4 drinks in male

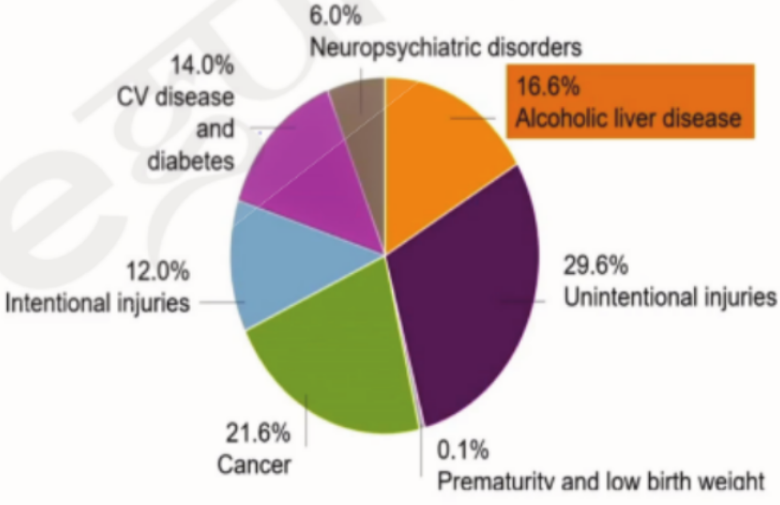
Arrack – 40-50% alcohol
Toddy – 5-10% alcohol

Males – 40-80 gm/day → for 10 yrs
Females – 20-40 gm/day – 10 yrs

⇓

Cirrhogenic.

10-20% heavy drinkers progress to cirrhosis



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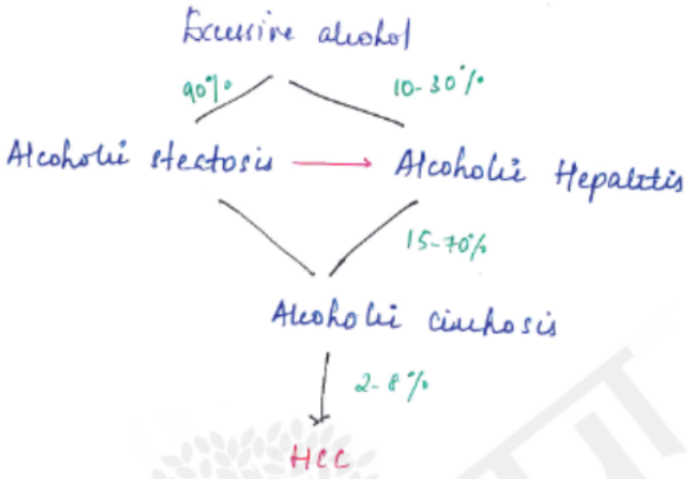

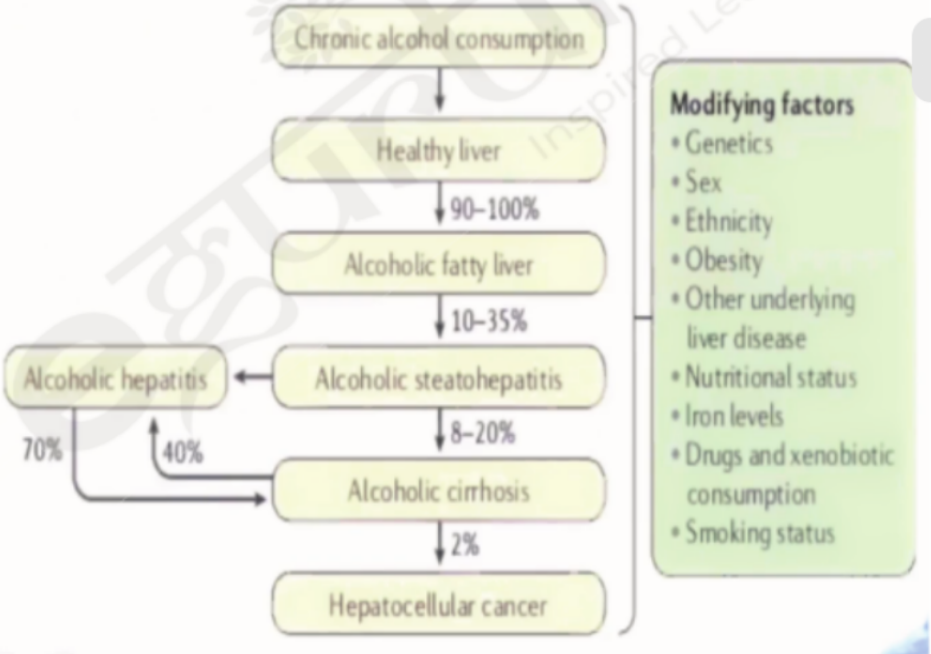

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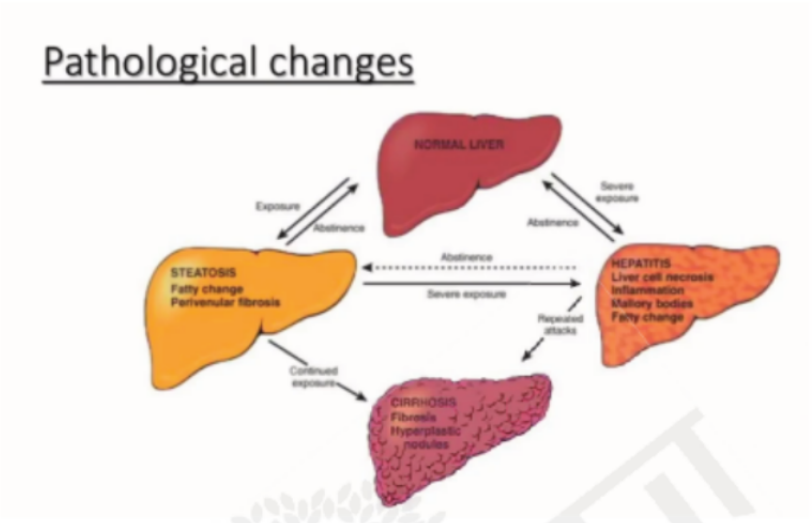
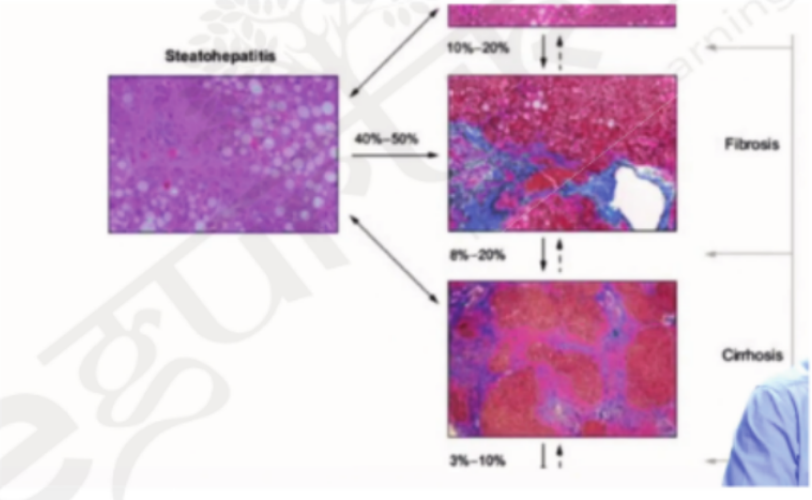
Pathological changes





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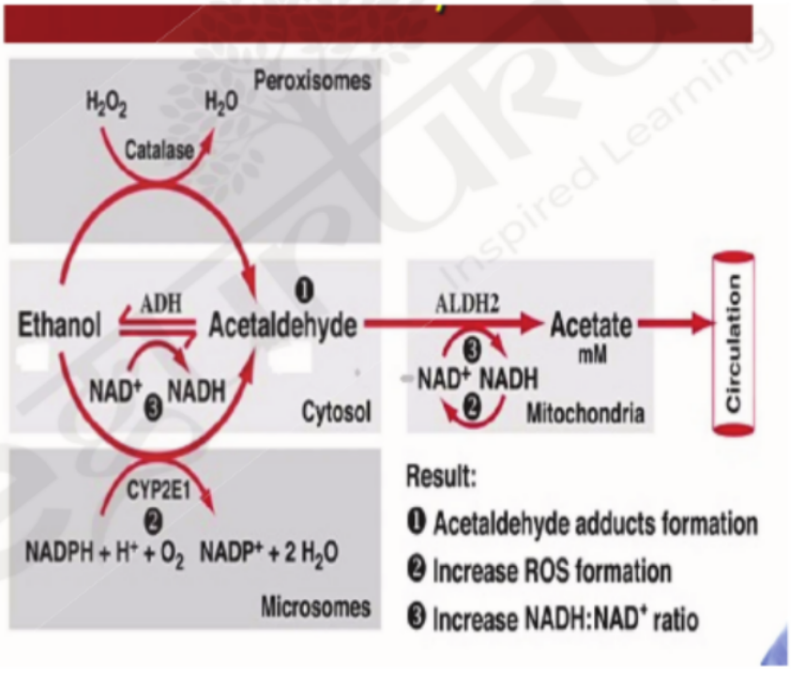
Alcohol is metabolized to acetaldehyde
It is toxic.

It makes Acetaldehyde adducts.

- ↓ Glutathione
- ↑ NADH : NAD ratio
- ↑ Free Radicals.



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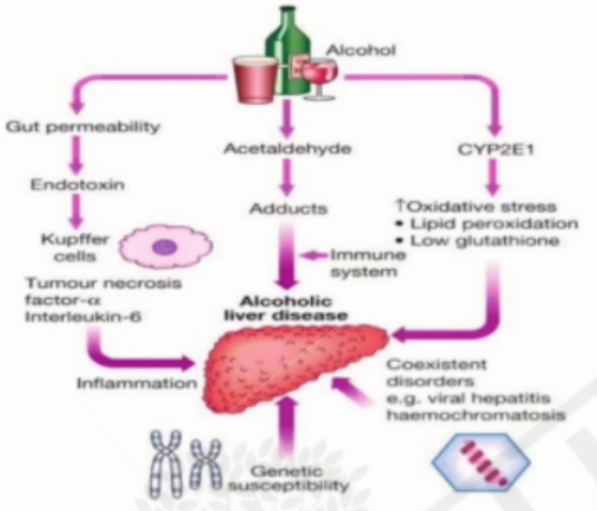

TABLE 335-1 Risk Factors for Alcoholic Liver Disease

RISK FACTOR	COMMENT
Quantity	In men, 40–80 g/d of ethanol produces fatty liver; 160 g/d for 10–20 years causes hepatitis or cirrhosis. Only 15% of alcoholics develop alcoholic liver disease.
Gender	Women exhibit increased susceptibility to alcoholic liver disease at amounts >20 g/d; two drinks per day is probably safe.
Hepatitis C	HCV infection concurrent with alcoholic liver disease is associated with younger age for severity, more advanced histology, and decreased survival.
Genetics	Patatin-like phospholipase domain-containing protein 3 (PNPLA3) has been associated with alcoholic cirrhosis.
Fatty liver	Alcohol injury does not require malnutrition, but obesity and nonalcoholic fatty liver are risk factors. Patients should receive vigorous attention to nutritional support.

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Clinical syndromes of Alcoholic Liver Diseases

Fatty Liver

- Asymptomatic
- Abnormal liver biochemistry
- Normal or large liver
- RUQ discomfort
- Nausea
- Rarely jaundice

Alcoholic hepatitis

- Asymptomatic
- Jaundice
- Malnutrition
- Hepatomegaly
- Features of portal hypertension e.g. ascites
- encephalopathy

Cirrhosis

- Stigmata of chronic liver diseases
- Ascites/varices/encephalopathy
- Large, normal or small liver
- Hepatocellular carcinoma

Investigations

LFT

→ AST, ALT } < 400 IU/L.

→ AST : ALT > 2:1

→ GGT ↑ → Not Most Accurate

→ Carbohydrate deficient transferrin (CDT)

→ MCV ↑ - Macrocytic

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Table 2
Sensitivity, specificity, positive predictive value, and negative predictive value of selected laboratory tests in the diagnosis of ALD

Laboratory Test	Relevant ALD Subtypes	Value	Comment
AST, ALT[46,49]	AFLD AAH Chronic ALD	Sn: 35%–50% Sp: 38%–86%	Limited sensitivity More accurate age 30–60
GGT[4,46,47,82–84]	AAH	Sn: 71%–73% Sp: 18%–75% PPV: 82%–92% NPV: 33%–40%	Helpful in early ALD Cheaper than CDT Increased by obesity
MCV[4,46,82,83]	AAH Chronic ALD	Sn: 52%–64% Sp: 66%–85% PPV: 73%–78% NPV: 70%–76%	Elevation may suggest long-standing ALD Insensitive in detecting relapse
CDT[4,46,81]	AAH Chronic ALD	Sn: 69%–88% Sp: 52%–97%	Limited or no use in some laboratories Expensive May help distinguish ALD from non-ALD Many false positive
Mitochondrial AST[48]	Chronic ALD	Sn: 93% NPV: 82%–100%	Limited or no use in some laboratories Good NPV
ETG[45,49]	AAH	Sn: near 100%	Present in all body fluids but urine easiest to test Very sensitive, so high false-positive rate because of inadvertent use of alcohol-containing products (ie, mouthwash, hand sanitizers)

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; CDT, carbohydrate-deficient transferrin; ETG, ethyl glucuronide; GGT, γ-glutamyltransferase; MCV, mean red cell volume; NPV, negative predictive value; PPV, positive predictive value; Sn, sensitivity; Sp, specificity.

22.17

Alcoholic Liver disease: Stanley Cohen

The best-studied of these tests, carbohydrate-deficient transferrin (CDT), correlates with alcohol use, and is based on the fact that chronic alcohol use reduces the number of carbohydrate moieties attached to transferrin.[45,46] CDT can therefore help distinguish ALD from non-ALD, but expense, false-positive rates, and variability of results based on age, weight, and gender limit it from wider use (see Table 2).[46,47,49] CDT is also affected by baseline serum transferrin levels, hypertension, and the presence of chronic viral hepatitis or decompensated liver disease.[50] CDT is also not sensitive for modest alcohol use, with data indicating a minimum threshold for detection of 60 g of alcohol daily for weeks.[45]

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Alcoholic Hepatitis

- Patient develop jaundice < in 60 days of heavy alcohol consumption.
- Serum bilirubin > 8 mg/dL
- AST : ALT > 1.5 ALT / AST | < 300

Clinical features

- Jaundice
- Fever
- Anorexia
- Tender Hepatomegaly.

AST / ALT | < 300 AST : ALT > 2.

Maddrey's Discriminant function

DF = 4.6 x (PT – control PT) + T. Bilirubin

DF > 32 → Severe Alcoholic Hepatitis

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Lille Score

- Age
- Creatinine
- Albumin
- PT
- Bilirubin

Lille score < 0.45 → Good survival (6 month) 85%

≥ 0.45 → " 25%

Glasgow Alcoholic Hepatitis score

↳ >9 → Mortality > 45%

Glasgow Alcoholic Hepatitis Score

Score	1	2	3
Age	< 50	> 50	
WCC (× 10^9/L)	< 15	> 15	> 2.0
Urea (mmol/L)	< 5	> 5	> 250
PT ratio	< 1.5	1.5-2.0	
Bilirubin (µmol/L)	< 125	125-250	

A score > 9 is associated with a 40% 28-day survival, compared to 80% for patients with a score < 9

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Model for End-stage Liver Disease (MELD)

	1-year survival (%)	
MELD score	**No complications**	**Complications**
< 9	97	90
10-19	90	85
20-29	70	65
30-39	70	50
MELD from SI units		
10 × (0.378[ln serum bilirubin (μmol/L) + 1.12[ln INR] + 0.957[ln serum creatinine (μmol/L)] + 0.643)		
MELD from non-SI units		
3.8 [ln serum bilirubin (mg/dL)] + 11.2 [ln INR] + 9.3 [ln serum creatinine (mg/dL)] + 6.4		

MELD Score

- S. Bilirubin
- INR.
- S. Creatinine

Rx

- Sustain alcohol abstinence.
- Supportive
  - Nutritional (thiamine)
  - Infections

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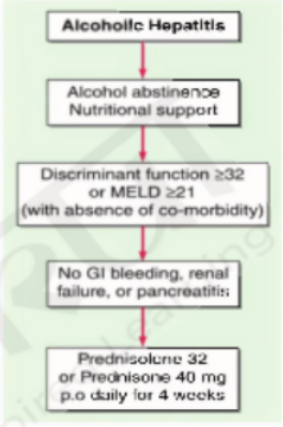
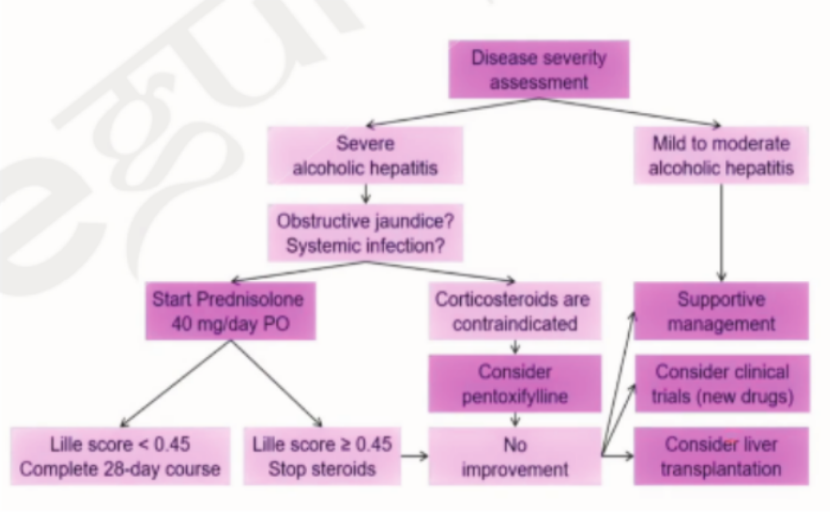
- GI bleed prophylaxis

- Pharmacotherapy
  - Steroids
  - Pentoxyphilline } Max. effect
  - NAC, Anti TNF.





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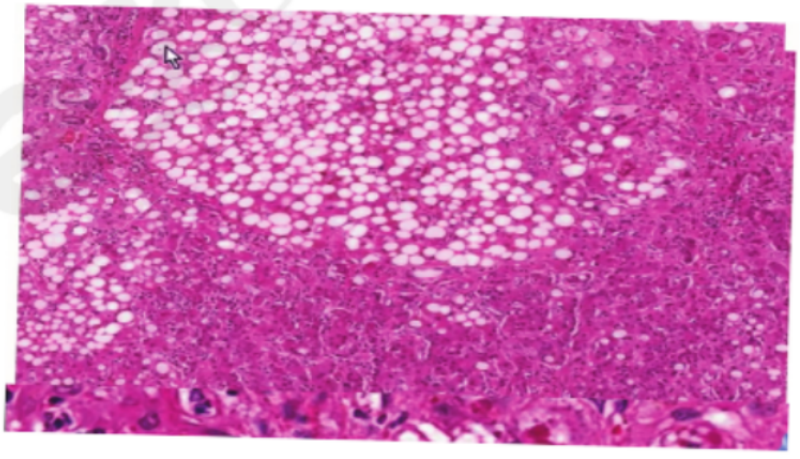
Alcohol Abuse vs. Alcohol Dependence

- Alcohol abusers as those who drink despite recurrent social, interpersonal, and legal problems
- Dependence - presence of 3 or more symptoms
  - a) tolerance
  - b) withdrawal symptoms
  - c) loss of control over drinking
  - d) strong desire to drink
  - e) drinking despite harm

- CAGE Questionnaire
- AUDIT Questionnaire

To check alcohol related problems.



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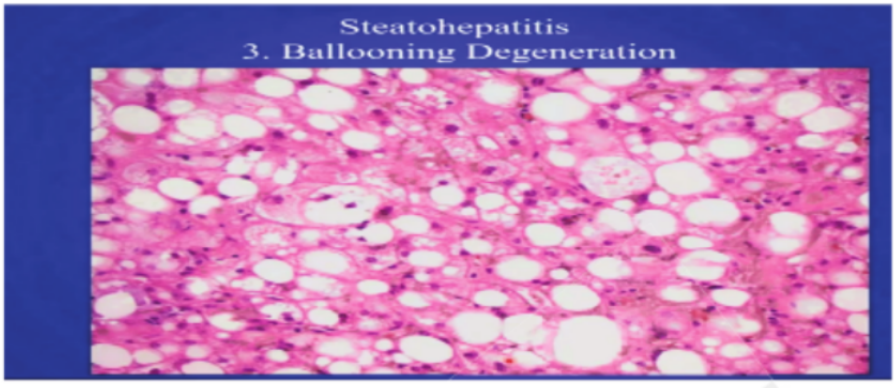

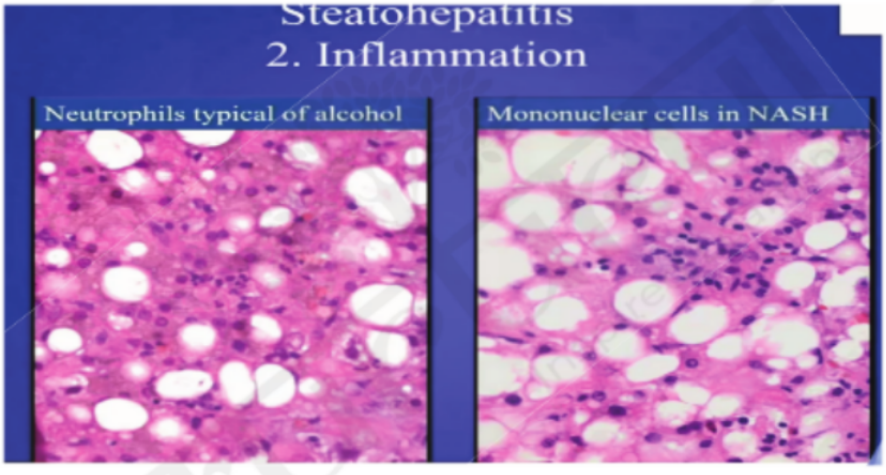

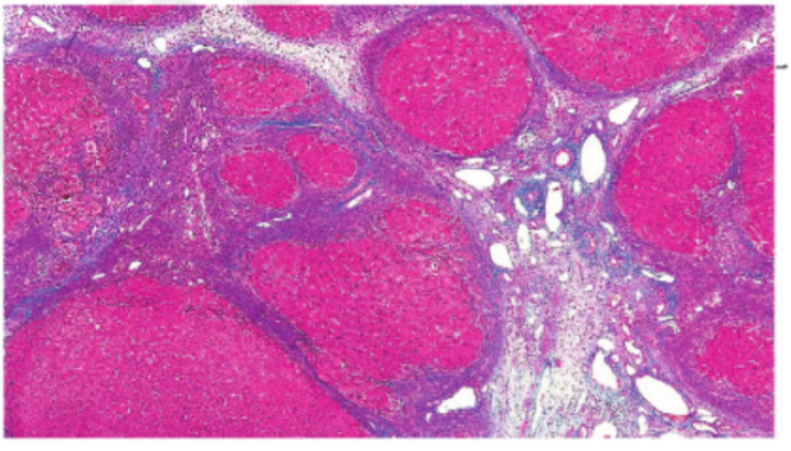

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Biological functions	Gene polymorphism (SNP)	Associated
Alcohol dependence	γ-Aminobutyric acid receptor subtypes (*GABRA2*)	Modified tolerance to withdrawal, aversion, positive reinforcement
Alcohol metabolism	Alcohol dehydrogenase (*ADH*) Acetaldehyde dehydrogenase (*ALDH2*2*)	Promotion of flush syndrome and reluctance to drink alcohol
Oxidative stress	Microsomal cytochrome P450 (*CYP2E1*) Glutathione S-transferase (*GST*) Manganese superoxide dismutase (*MnSOD*)	Impaired detoxification, aggravated hepatocyte injury, oncogenic pathway stimulation
Endotoxin pathways	Endotoxin receptor (*CD14*) Tumor necrosis factor-α (*TNFA*) Interleukin-10 (*IL10*)	Altered pathogen and damage associated pattern recognition, enhanced liver inflammation
Lipid storage and utilization	Adiponutrin (*PNPLA3*) Peroxisome proliferator-activated receptor γ (*PPARG*) Apolipoprotein A (*APOE*)	Altered rates of fatty acid oxidation, de novo lipogenesis lipid export

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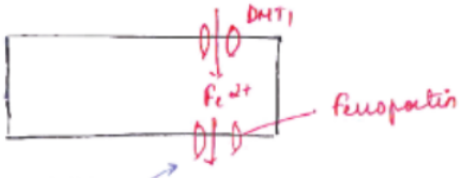
HEMOCHROMATOSIS

Hereditary Hemochromatosis

- Excessive Iron Absorption from Intestine

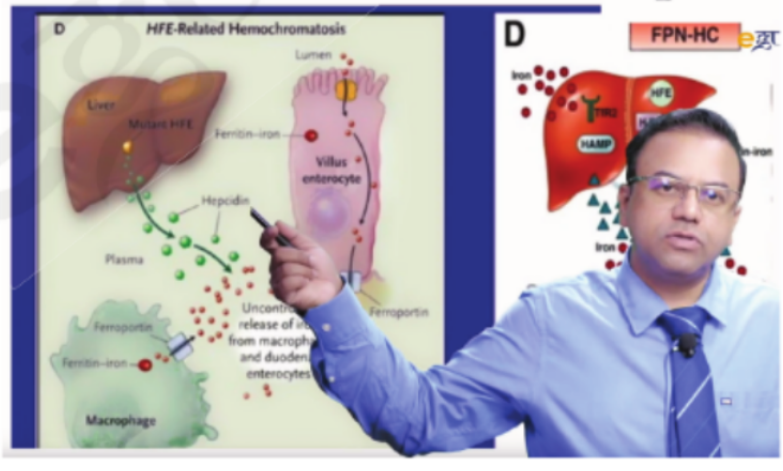


→ Carrier protein.

Liver → Hepcidin

Low Hepcidin

- HFE mutation — H/C cause
- Hemojuvelin
- Tf - B - 2



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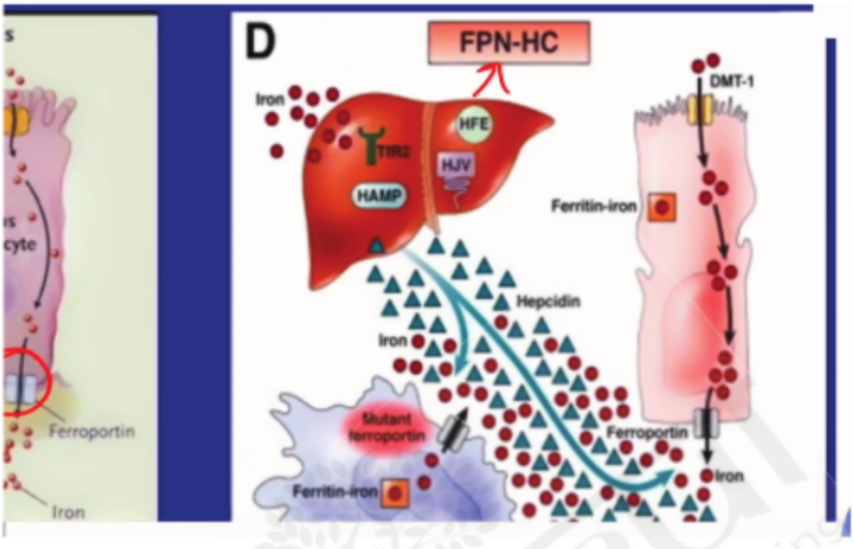

Subtypes	Defect	
1) classic Hemochromatosis (80%)	HFE	AR
2) Juvenile	2A: Hemojuvelin	AR
	2B: Hepcidin	
3) Type-3 Hemochromatosis	Tf - R - 2	AR.
4) Ferroportin disease Hemochromatosis	Ferroportin	AD

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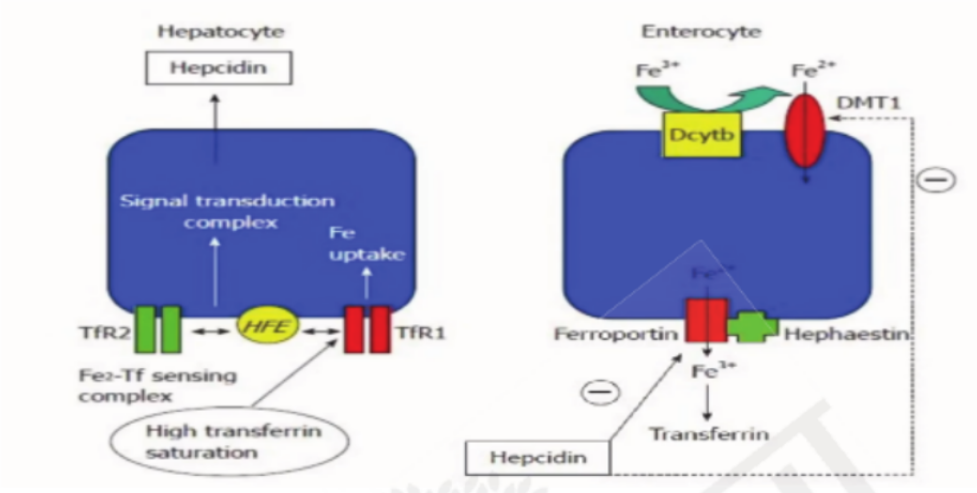

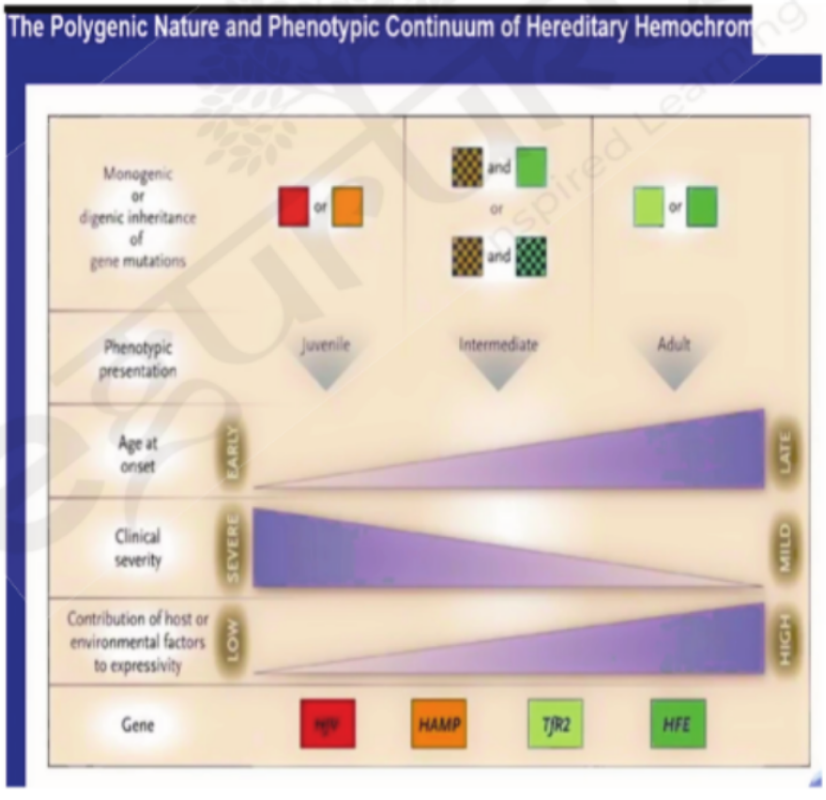

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	Juvenile	HFE
Age	< 30 yrs	> 30 yrs
M:F	1:1	F > M (1:2 – 1:10)
Severity	+++	+

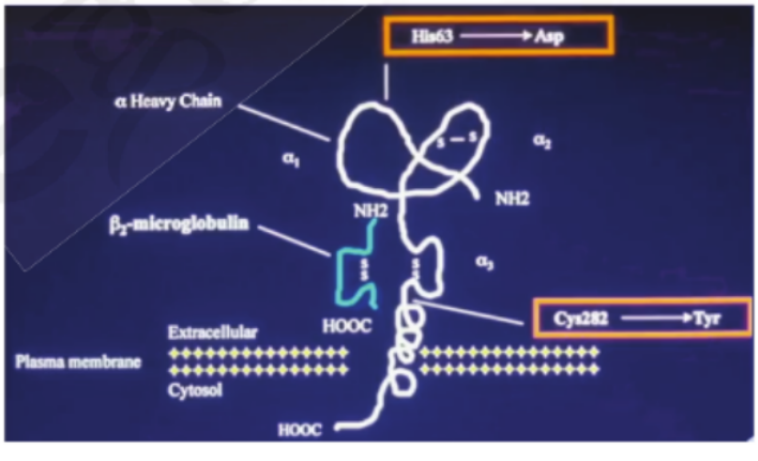
M/C Mutation – HFE (protein)

Sub mutations

Cys - 282
H63D
] Mutations

- HLA - A3 associated disease
- Defect occur in chr. 6.

HFE Protien



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C/F

- 21% Pts - symptomatic
- ↑ LFT's
- Fatigue
- Pigmentation - skin - ↑ melanin } slate grey skin.
  - Iron deposition
- Diabetes (50% Pts) (Bronze)
- Arthralgia / Arthritis → MCP* - 2nd/3rd
  - osteoarthritis*
  - wrist - pseudogout
  - knee
  - Ankle.
- Hypogonadism - pituitary. (2°)
- Cardiomyopathy → Arrythmias - 23%
  - Restrictive
  - & Dilated
- Thyroid disease
- Infections

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- Liver dysfunction - common
  ↓
- Hepatitis - Fibrosis - cirrhosis
  - Decompensated
  - HCC.

- Hereditary Hemochromatosis - 20 fold ↑
  ↓
  HCC.

Screening

- Symptomatic pts.
- Asymptomatic - ↑ Iron stores.
- 1st degree relative - Hereditary Hemochromatosis

IOC → Transferrin saturation (>45%)
(screening)

- Ferritin levels > 200 - Males.
  > 150 - females.

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Genetic testing

- Cys-282
- H63D.

Transferrin saturation >60% - Male
> 50% - Female
] 90% Accuracy

↓

Hereditary Hemochromatosis

Transferrin saturation <45% → 97% Negative predictive value.

Ferritin level >1000 → Risk → Fibrosis.

Liver Biopsy

Hepatic Iron Index

$$= \frac{HIC}{Age} \geq 1.9$$

↓

Hereditary Hemochromatosis.

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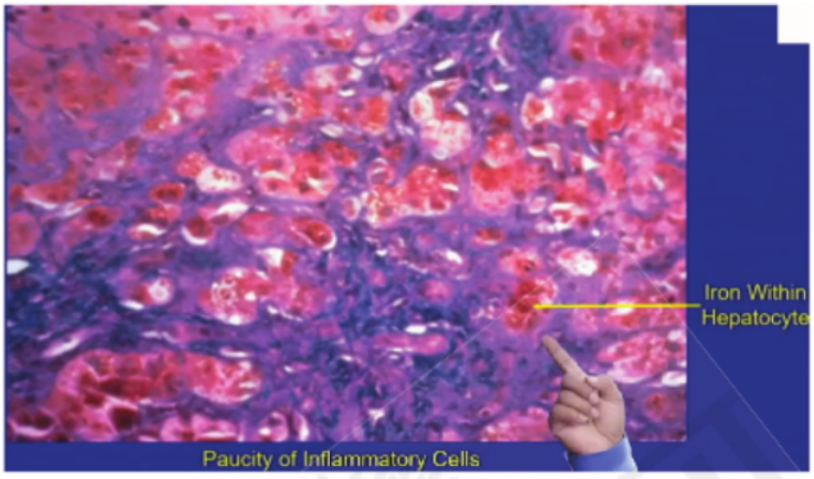

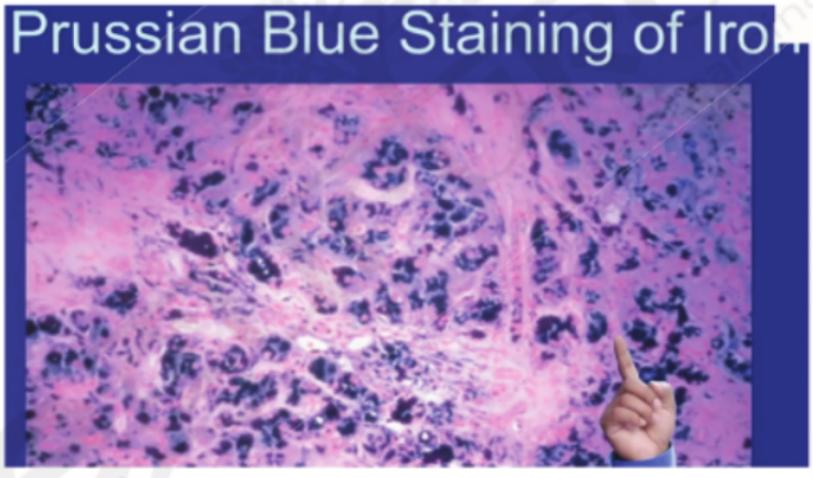

Treatment

- Phlebotomy

Goal → Trying to get ferritin level 50 – 100 μg/L.

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- Cirrhosis
- Testicular atrophy
- Arthropathy

} No benefit of phlebotomy & reducing ferritin level. No exercise.

- ↑ Energy level
- ↑ cardiac function
- ↑ control of DM.
- ↓ Abd pain
- ↓ portal pressure

} Benefits

How to treat with Phlebotomy

- Each unit of blood contains 200-250mg of iron.
  - Normal total body iron is 2-6g. HH >30g
- Start with one unit per week. Check ferritin and TS after every 12 units.
  - Check more frequently as you approach target ferritin.
- Hgb and Hct before each phlebotomy (avoid decreasing below 80% baseline).
- Once target reached, monitor ferritin, TS to determine frequency of phlebotomy.

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Dietary Intake

- Limit → Vit C ↓
- Avoid Iron supplements
- Avoid alcohol

- Chelation Therapy
  - Deferiprone – oral
  - Deferasirox } parenteral
  - Desferoxamine }

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WILSON'S DISEASE

- Autosomal Recessive
- Defect in Chr. 13
- ATP 7B transporter defect. (for copper)

Copper

- Absorption – 1-2 mg / day
- Requirement – 0.75 mg / day.

Excretion

- Biliary
- 10-15% – renal loss.

Major function of ATP 7B transporter

- Transport of copper for excretion of bile.
- Allows incorporation of copper in ceruloplasmin.

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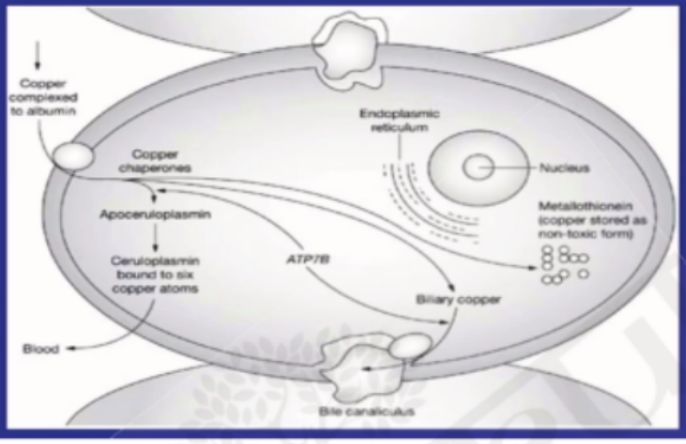
Intracellular Copper Transport



Core problem → Hepato lenticular degeneration
(liver, brain)

- Wilsons – 1/30,000 population
- ATP 7B – >500 Mutation

Diagnosis

- 5-35 years

Young – hepatic manifestations

Older – Neuropsychiatric manifestation.

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→ Liver dysfunction

– Neurologic
  - Basal ganglia
  - Frontal
  - Brainstem

– Psychiatric symptoms

Liver

Acute Hepatitis → chronic hepatitis → Cirrhosis

Decompensated

HCC

Spectrum

– Alk. Phos are – ↓

– Acute liver failure

Transaminases < 1500 (2000)

AST : ALT > 2.2.

Alk. Phos : Bilirubin – < 4

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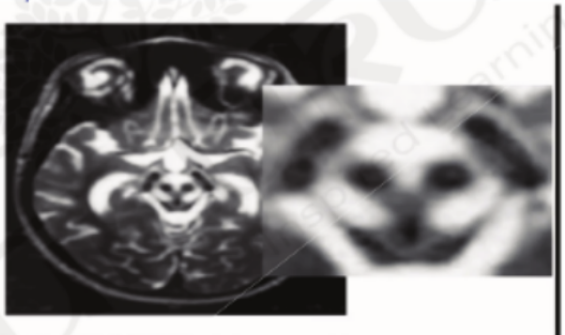
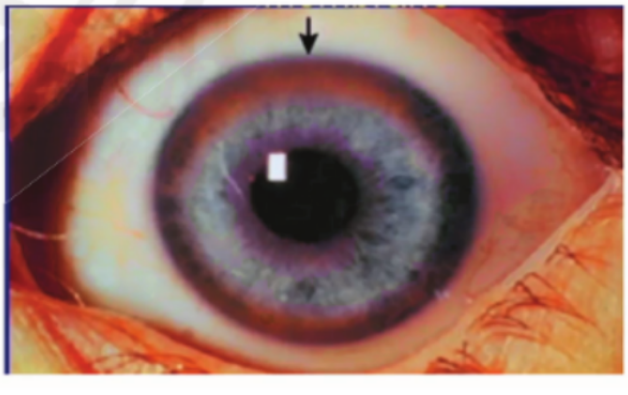
Neurological

- Dysarthric
- Gait Ab- (N) / Ataxic
- Dystonic
- Tremor → Wing beating
- Parkinsonism
- Drooling



Hyper intensity from neuron loss, gliosis and associated increase in water content

Das SK and Ray K (2006) Wilson's disease: an update
Nat Clin Pract Neurol **2**: 482–493 10.1038/ncpneuro0291



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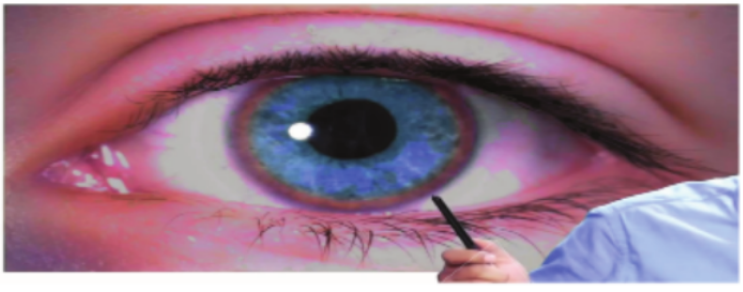

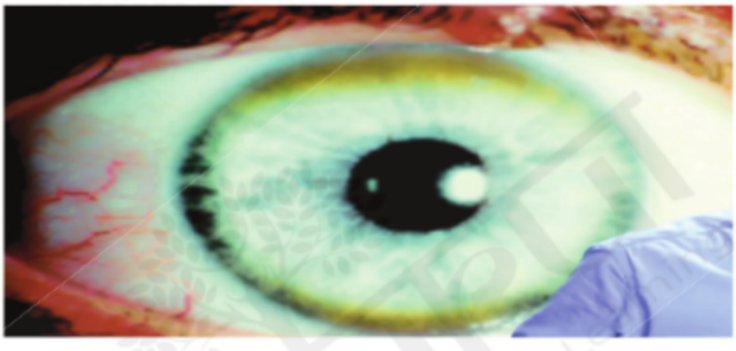

Kayser Fleischer

- Slit lamp
- Neuro - > 80%
- Hepatic - 30 - 50%
- Sunflower cataract
- Type - 2 RTA
  - ↳ proximal tubule defect
- Bone - fracture ↑

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- Blood → Coomb's negative Hemolytic Anemia.

Investigation

1) Slit lamp → Kayser Fleischer
2) S. ceruloplasmin ↓
3) 24 hr urine copper.
   > 100 µg/day
   > 40 µg/day → Investigate for wilsons
   ⇓
   Penicillamine challenge.
   ↓
   Excretes urine copper (↑)

→ S. copper (Total) – Low.

→ Free copper – ↑

→ Hepatic copper – ↑ (gold standard)

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Liver Biopsy

>250 µg/g of liver → dry weight
↓
confirms Dx.

<50 µg/g → exclude WD.

Treatment

- Penicillamine – can worsen neurologic Sx
- Trientene – mobilise accumulated Cu to urine
- Tetrathiomolybdate → Neurodysfn.
- Zinc. → (Best choice).
  ↳ prevents absorption of copper in intestine.
  → Maintenance
  – Asymptomatic

Trientene & Zinc → Not given at same time
(At least 2 hr gap)

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Markers for Cu deficiency

- Leucopenia
- Anemia.

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PBC & PSC

Primary Biliary cholangiopathy

- M/c - Autoimmune liver disease.
- 90% - females.
- Median age - 4th/5th decade.
- 40 - 400 / million

Presentation

- Asymptomatic
  - Alk. Pos are ↑

S/P

- Pruritus
- Fatigue

Diagnostic criteria

- 2/3 should be fulfilled
- Alk. Pos ↑ > 1.5 x ULN
- Anti mitochondrial Ab +ve (seen in 90% of pts)

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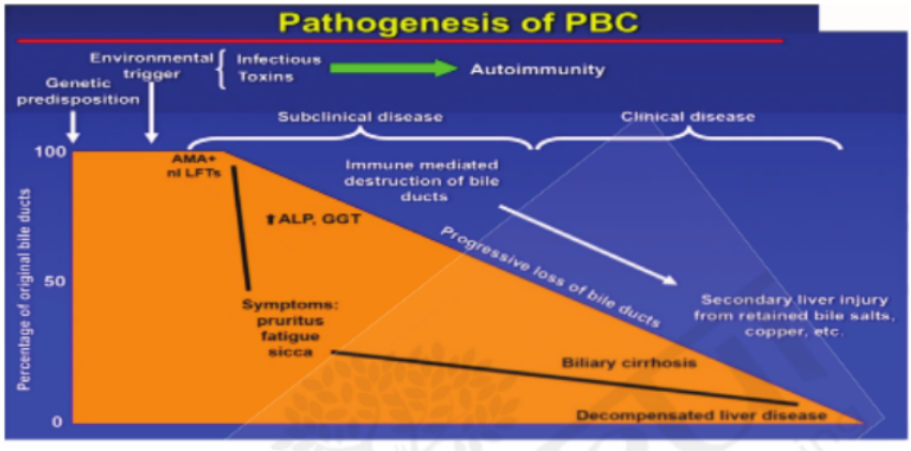
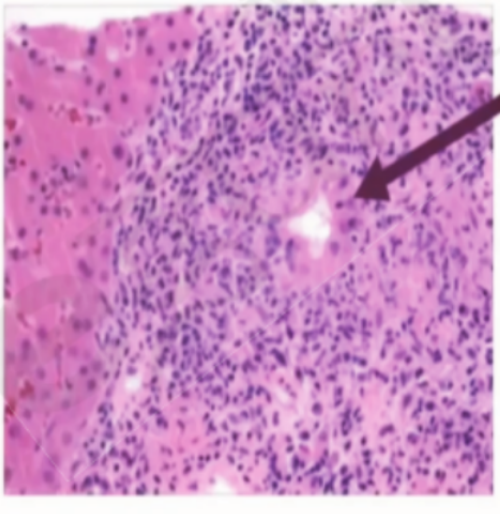
– Liver biopsy





- Florid duct lesion
  - Inflammation
  - Injury to bile duct epithelium
  - Disruption of bile duct basement membrane
- Granulomas
- Ductopenia

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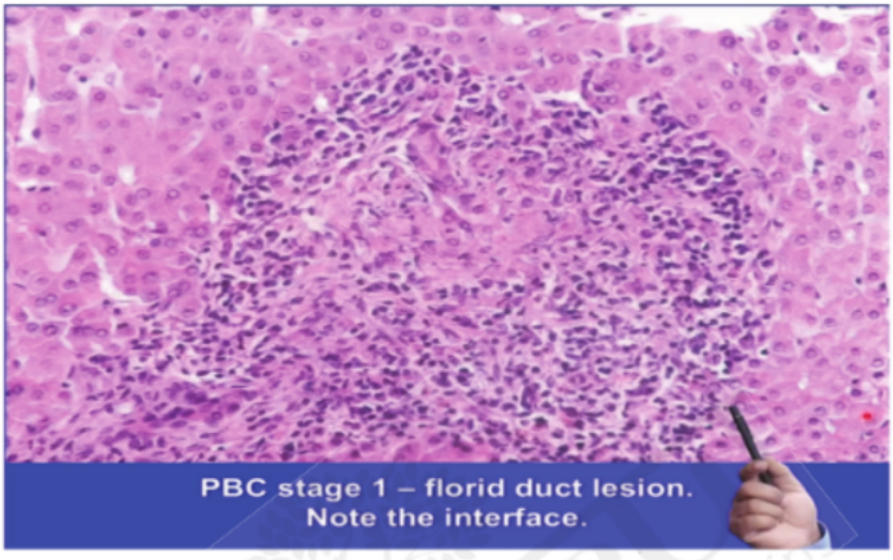

Clinical Variants

1) AMA -ve PBC

2) PBC – AI hepatitis → Overlap Sx.

- ALT ↑
- Ig G
- Anti smooth muscle Ab.

3) Diagnosis

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Predictive Factors for Death or Transplantation in UDCA Treated Patients: Paris 1 Criteria

Table 5. Multivariate Analysis of Predictive Factors for Death or Liver Transplantation in UDCA-Treated Patients

Variable	*P*	Relative Risk (95% CI)
Total serum bilirubin >1 mg/dL*	0.0131	1.7 (1.1-2.6)
Histological stage 3-4*	0.0445	1.5 (1.0-2.2)
Moderate to severe interface hepatitis*	0.0022	1.9 (1.2-2.9)
Absence of biochemical response at 1 year according to the present study's criteria†	<0.0001	2.3 (1.5-3.7)

*At the onset of treatment. †Total serum bilirubin >1 mg/dL, or ALP >3 ULN, or AST >2 ULN.

Corpechot C, Hepatology 2008;48:871

Rx

1st line → UDCA
(ursodeoxycholic Acid)
- 13-15 mg/kg/day

Symptoms (pruritus)
- cholestyramine, sertraline, naltrexone, rifampin

Ca^{2+} & vit D.

40% pts → Do not respond to UDCA.
↳ Obeticholic Acid (OCA)
↓
pruritus occurs.

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Obeticholic Acid
- 5mg/day — Non-cirrhotic Child Pugh class A.

5mg/week — child Pugh class B/C.

- Benzafibrate
- Fenofibrate

10% pt → Need liver transplant.

Primary sclerosing cholangitis (PSC)

- Chronic progressive, cholestatic liver disease c̄ unknown etiology.
- Inflammation + Fibrosis
  - Extrahepatic biliary tree
  - Intrahepatic biliary tree.

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Male — More common.

Age - 30 – 40 years.

Association c̄ — Inflammatory Bowel Disease.

— ® colon more involved.

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CF

- Asymptomatic - pruritus → cholangitis
- ↑ Alk. Pos are
- AB → P-ANCA +.
- 10% - IgG-4.

cholestatic pattern

Alk. Pos ↑↑

↓

① USG - 1° Investigation

② AMA

IBD

↓

MRCP

↓

Sclerosing cholangitis.

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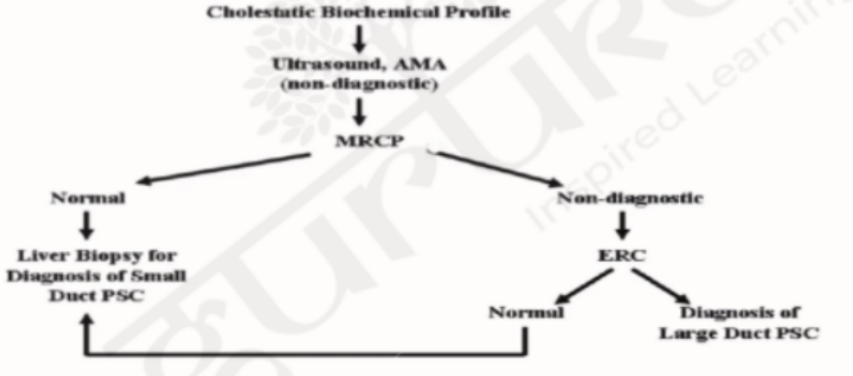
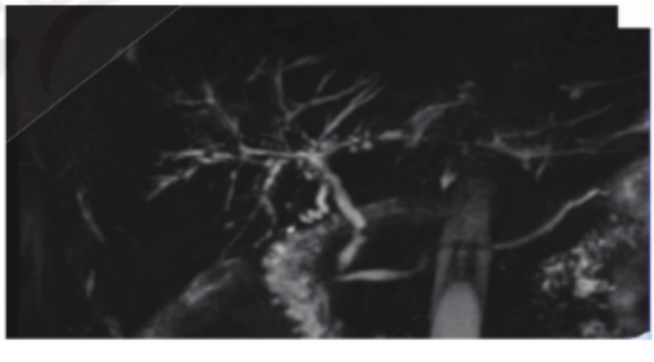
Rule out Secondary Causes of Sclerosing Cholangitis

AIDS cholangiopathy
Cholangiocarcinoma
Choledocholithiasis
Diffuse intrahepatic metastasis
Eosinophilic cholangitis
Hepatic inflammatory pseudotumor
Histocytosis X
IgG4-associated cholangitis
Intra-arterial chemotherapy
Ischemic cholangitis
Mast cell cholangiopathy
Portal hypertensive biliopathy
Recurrent pancreatitis
Recurrent pyogenic cholangitis
Surgical biliary trauma





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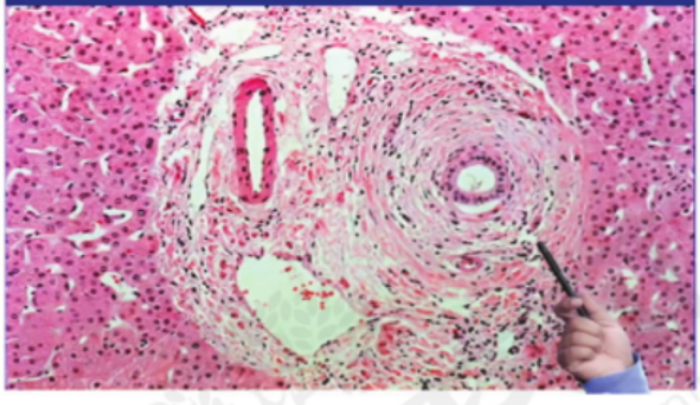
Onion skin pattern 24.40

PSC: Histology



Rx

- No Medical Therapy
- UDCA

Dose g/kg/d	17-23 mg/kg/d	25-30 mg/kg
ts improve	Liver tests improve	More treatment fai
ges in survival	Trend toward improved survival	Death, liver transpl variceal bleeding, hi rates of colorectal

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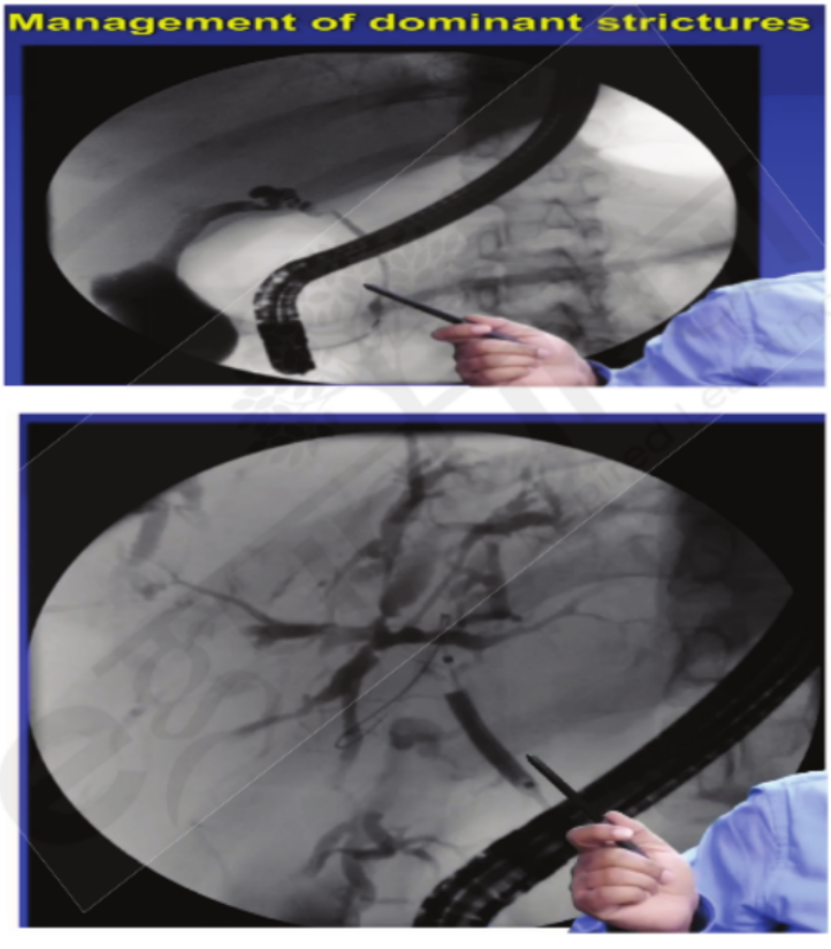
- Balloon - dilatation
- Stenting - Not good option



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Complications

- cholangiocarcinoma.
- Bacterial cholangitis

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